THE EVALUATION OF THE CHANGES IN LIPID LIPOPROTEIN PROFILE INDUCED AFTER INGESTION OF SINGLE HIGH CHOLESTEROL TEST DIET

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This is to certify that the work entitled "THE EVALUATION OF THE CHANGES IN LIPID LIPOPROTEIN PROFILE INDUCED AFTER INGESTION OF SINGLE CHOLESTEROL TEST DIET", which is being submitted as thesis for M.D. (Medicine) examination of Dundelkhand University, 1989, by Dr. Rajeev Mangal has been carried out under my guidance and supervision. The techniques and statistical methods used, were undertaken by the candidate himself and were periodically checked.

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INTRODUCTION

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Atherogenesis results due to interaction of multiple factors viz. age, smoking, hypertension and hyperlipidemia. Diet also plays a vital role in its causation. Modification of diet has led to progression or regression of atherosclerotic lesions in several experimental models (John et al. 1982). However individual response to high cholesterol fat challenge varies enormously but remains constant for an individual over a long period of time (Kingsbury K.J. 1960).

Importance of basal fasting cholesterol level in calculating an individual risk for coronary artery disease has perhaps been over emphasised. Over more than forty percent of young patients of documented CAD do not reveal raised fasting cholesterol level (Gregory S. et al. 1983), yet they have rampant atherogenous vascular involvement. What is the mechanism of atherosclerosis in such cases?

Zilversmit in 1973 postulated that atherogenesis may be a post prandial phenomenon. Transient post prandial

rise of Beta VLDL, chylomicron and formation of several species of unusual lipoproteins, may cause repeated cholesterol deposition in cells in arterial wall over the years. While fasting cholesterol value may remain well within normal range over the same duration.

Thus it seems logical, that post prandial response of an individual to high cholesterol fat load may more appropriately be related to his risk of developing atherosclerosis in future. Little has been done in establishing a correlation between post prandial response of an individual and risk of atherogenesis. Even different post parandial responses after cholesterol fat load have not been studied in depth. Proper defination of these responses and correct interpretation may perhaps be the first major step towards formulation of cholesterol tolerance test.

Previous effort in this directions by several after workers (Albrink and Man 1956, Pomerenze, 1954) all over the world have borne little fruits, primarily because each of them calculated serum total

cholesterol and other lipid subfraction 2-6 hour after test load, thinking that cholesterol is a slowly absorped substance and can not alter blood cholesterol level before two hours.

The identification of LDL receptors by Joseph Goldstein, and its role in control of serum cholesterol metabolism, has changed whole of the scenario. Cholesterol now no more remains such a inert substance as thought before, in fact disappearance of intravenous radio active cholesterol within 20 minuts of injection from the vascular compartment, reflects its high dynamic state with the tissue cholesterol. Perhaps this dynamic equilibrium is achieved by the presence of LDL receptor and yet undefined hormonal or neurogenic reflexes affecting these receptors.

As already stated, there is enormous individual variability in responses after cholesterol fat feeding. Results of previous studies in our department have further strengthen this view. Feeding high cholesterol fat breakfast for seven days in young and old subjects resulted in increased levels of serum total cholesterol,

with predominant rise of HDL in youngs and that of LDL in older subjects (Arora R.C., Gupta Gulab et al., 1984, Arora R.C., Gupta Vinod 1985). This study also brought out the fact, that in some subjects, prolong cholesterol fat feeding results in fall in STC level instead of rising. Present knowledge offers no satisfactory explanation for the above phenomenon.

Since prolong feeding is not practicable on a mass scale for screening purpose, so we decided to study changes in basal lipoprotein profile after ingestion of single high cholesterol load in normal and diseased human volunteers.

AIMS OF THE STUDY

1) To assess the changes in basal lipoprotein profile after giving single high cholesterol test load.

2) To correlate these changes for quantitative and qualitative risk of an individual for atherogenesis.

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REVIEW OF LITERATURE

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Numerous studies have implicated altered levels of plasma lipoprotiens in the pathogenesis of atherosclerosis. In particular, elevated low density lipoprotein (LDL) and, diminished high density lipoprotein (HDL) cholesterol levels appear to be strong risk factors for the development of atherosclerosis.

Research of the last two decades has revealed a rather complex set of events that control plasma lipoprotein level. Specific proteins have been implicated in the regulation of lipoprotein synthesis. Beside this many more factors like age, sex, cigarette smoking, obesity, hypertension, dietary habits and sedentary life style exert their influence on lipoprotein levels and development of atherosclerosis in their own way. Many of the risk factors are reversible but influence of age, sex and genetic factors are irreversible.

Atherosclerosis is essentially a degenerative process associated with advancing years, mainly affecting larger arteries, particularly the coronary and cerebrals.

The lesion of atherosclerosis to start with is a fatty streak. This lesion can be found in majority of children of 10 years of age. Second stage in advancing atherosclerosis is a fibrous plaque and an finally it gives way to advanced lesion.

CHANGES IN LIPID LIPOPROTEIN LEVELS AFTER HIGH CHOLESTEROL DIET

Effect of long term and short term feeding of diet rich in cholesterol evokes variable responses and is subject to individual variation. Dietary fat and cholesterol causes changes in specific lipoprotein in a variety of species(Mahley et al, Arora R.C. et al). Quantitatively, a change in specific lipoprotein may be dramatic in one species than in another. These changes have been associated with the development of atherosclerosis in experimental models (Mahley et al). Considering effect of diet on individual lipoprotein fractions.

TOTAL SERUM CHOLESTEROL (STC)

In 1956 Ancelkeys, J.T. Anderson et al concluded that serum cholesterol level is essentially independent of the cholesterol intake over the whole range of natural human diets. But later on it was

proved beyond doubt that feeding cholesterol rich diet for 2-8 weeks raises total serum cholesterol in blood (Arora R.C. et al., Messinger et al., Conner et al., Deborah Applebaum et al.).

In an earlier report, Bruhn in 1940 observed a 20% rise in mean cholesterol level after a fat load. Effect of high cholesteral fat load on post prandial cholesterol levels has also been studied in the past by several workers, but insignificant difference has been found between post prandial and 10 to 14 hrs. fasting value (Albrink and Man 1956, Pomeranze et al. 1954, Schilling et al. 1964). All these workers observed plasma cholesterol values up to 24 hrs. after a test meal. On the other hand Mikkila and Konttinen in 1962 demonstrated a significant decrease in cholesterol level six hours after a fat diet in healthy soldiers.

Hanno Krauss, Pieter Groot in Oct. 1967 reported insignificant changes in total serum cholesterol after feeding 0.5 gm/m^2 of cholesterol and taking readings at 2 hourly interval for 14 hrs.

In adolescents with initial cholesterol levels greater than 200 mg/dl, a 30 percent decrease in cholesterol

intake led to an appreciable drop (15.6%) in cholesterol levels, but the effect was much more modest (8.3%) in those with lower initial levels (M.C. Gandey et al. 1972).

In another large survey of school children, there was no positive correlation between the low (80-130 mg/dl), the intermediate (157 to 180 mg/dl) and the high (194 to 426 mg/dl) cholesterol levels, with the mean daily intake of energy, sugar, fat, saturated fat and cholesterol (Weidman et al. 1978). However, in 7 different studies summarised recently, significantly weak, correlations were noted between serum lipids and dietary P/S ratio (Mellies and Glueck 1983).

In a survey of school age children examining the influence of nutrients on LDL cholesterol, it was concluded that the higher intake of cholesterol and lower ratio of P/S was associated with higher value of LDL cholesterol. Strict vegetarians have been reported having lower serum cholesterol than lactovegetarians and nonvegetarians (Sacks F.M. et al, 1975, Kmuiman J.T. et al, 1982).

Textured vegetable proteins lowered total serum cholesterol in hypercholesterolemic subjects with no change or a slight elevation of in HDL cholesterol, no effect or only minor changes have been observed in normolipidemic subjects (Sirtori C.R. et al., 1985).

The replacement of animal protein with vegetable protein in the diet has been suggested to reduce the diet linked atherogenic risk (Carroll K.K. 1982).

However, Sacks F.M. et al. 1983 found no appriciable correlation between total intake of protein, when consumed above minimum requirement and serum cholesterol level.

Work in animals showing that sucrose and fructose are atherogenic, prompted human studies, which have not shown consistent changes. In one study, isocalorie replacement of starch with sucrose in mixed diet did not lead to changes in serum cholesterol (Mann and Truswell, 1972), changes that were documented in another study (Reiser et al., 1978).

Early fat intake does not influence subsequent serum lipids level. Serum cholesterol was higher during the first 9-12 months of life in breast feed babies, but there was little difference subsequently (Friedman and Coldberg 1976, Huttunen et al. 1983). In addition to its high cholesterol content (20 mg/dl), breast milk has decreased P/S ratio of fatty acids when compared to formulas. In a report dealing with feeding habits and serum lipids in infants and children, there was a direct correlation between serum lipids and the amount of saturated fat as well its P/S ratio in infants aged 6 to 10 month, but no such correlation was found in 3-4 years old children. The type and duration of early feeding practices had little influence on subsequent serum lipid levels (Anderson et al. 1979). Results of human studies, therefore do not agree with animals work, which suggest that a low post natal dietary cholesterol homeostasis. In fact it was shown that children aged 7 to 12 years who were fed low cholesterol formulas had a lower mean serum cholesterol than those fed cow's milk or breast milk (Hodgson et al, 1976). Another study could not document an effect of a low versus a moderate cholesterol intake during first six month of life in response to large cholesterol

intake during second 6 month of life (Glueck 1972).

HIGH DENSITY LIPOPROTEIN (HDL)

High density lipoprotein are lipid-protein complexes defined by flotation in the ultra centrifuge between density 1.063 and 1.21 gm per ml, by the presence of major protein constituents, apolipoprotein A-I and A-II, and by alpha migration on electrophoresis. Three classes of HDL are seperated on the basis of flotation rates on ultracentrifugation. HDL_3 have flotation rates between 0-3.5, HDL_2 have rates in excess of 3.5. The third and minor HDL_1 is sometimes found at d $\geq$1.063 and overlaps with the low density lipoprotein distribution. Recently Mahley and Colleagues have identified a distinct sub type of HDL designated HDL_c or apo E-HDL_c. This is found in the plasma of cholesterol fed animals, and to a much smaller extent in humans fed high cholesterol, high saturated fat diets. HDL_c differs from other sub type by presence of apolipoprotein E. This property confers an affinity for the low density lipoprotein receptor (Mahley and R.W. Weisgraber, 1978).

The lipid constituents of HDL exhibit variations. Cholesteryl ester content may range from 10-20 percent,

Triglycerides are normally less than 4 percent. The ratio of cholesterol to triglyceride in HDL may show wide fluctuations with increase being observed after dietary cholesterol supplementation (Mistry P. et al., 1977) and decrease being found in patients with hyper triglyceridemia (Weisweiler P. et al., 1977), uremia (Brunzell J.D. et al., 1977), and Ischemic heart disease (Carlson L.A. et al. 1975).

The bulk of HDL mass appears to arise from the interaction of precursor particle nascent HDL secreted by the liver and intestines, with lipids and protein released during the catabolism of triglyceride rich lipoprotein. A portion of HDL also arises from transfer and uptake of lipids, particularly free cholesterol from cell membrane.

FACTORS MODULATING HDL LEVELS IN HUMANS

(a) Constitutional Factors

In most population it has been demonstrated that women have higher levels of HDL than men at all ages following puberty. Exogenous adrogen administration lowers HDL levels in men (Furman, R.H. et al., 1967). A drop in HDL level seen in males at around the time of puberty (Beagtehole et al., 1980) has been related to the degree of

sexual maturation. (Frerich R.R. et al., 1978 and Morrison J.A. et al., 1979).

Transient increase in HDL_2 have been reported at or near the time of ovulation. (Barclay M. et al., 1965). No changes in HDL cholesterol have been found during pregnancy (Kinnunen P.J. et al., 1980).

There also exist a strong genetic influence in disease states. Reduced levels of HDL cholesterol is found in adult first degree relatives and prepubertal and pubertal children of patients with a history of Acute myocardial infarction (Micheli H. et al., 1979, Pometta D. et al., 1979, Robertson F.W. et al., 1980). Recently, evidence for autosomal dominant inheritance of low HDL levels has been reported in large kindred with a high prevalence of coronary disease (Vergnaie C. et al., 1981). High level of HDL has also been reported in black American population (Tyroler et al., 1975).

HDL level also change with age. In males the levels are stable until puberty and adolescence, during which there is a decline followed by relatively stable levels in adulthood until ages 55-60, where there is an increase; and then a plateau in older age group. In females there is a small linear increase in

HDL-c from childhood to about 60 years, after which no age effect is apparent (Heiss et al., 1980).

(b) OBESITY AND HDL

HDL levels are lower in obese individual than in non obese controls (Wilson D.E. et al., 1972, Carlson L.A. et al., 1975 and Glueck C.J. et al.). During the course of weigh loss, an increase in HDL cholesterol concentration has been reported to occur in association with reduction in VLDL and total triglyceride concentration (Wilson D.E. et al., 1972). But in other studies HDL cholesterol showed either no change or a reduction (Widholm K. et al., 1978, Thompson P.D., 1979, Howard B.V., 1979).

(c) PHYSICAL ACTIVITY AND HDL CHOLESTEROL

High levels of HDL-c are reported to be related with high level of endurance type exercise, including long distance runners, cross country skiers, lumberjacks, tennis player, and soccer player (Wood P.D. et al., 1977, Lehtonen A. et al., 1978, Lehtonen A. and Viikari et al., 1978, Vodak P.A. et al., 1980).

Reduction in adiposity, in combination with mild exercise program, resulted in no increase in HDL cholesterol, whereas a drop in HDL cholesterol

was found with caloric restriction in the absence of exercise (Weltman et al., 1980).

(d) ALCOHOL AND HDL

Alcohol ingestion has been reported to raise levels of HDL (Johansson B.G. et al., 1974, Belfrage et al., 1977). But the results of Gluck C.J. et al., 1980 were contradictory to the above statement.

In a large epidemiological study levels of HDL cholesterol and amount of habitual alcohol intake in moderate range have been independently correlated (Castelli W.P. et al., 1977).

(e) RELATIONSHIP OF DIET AND HDL CHOLESTROL

Diet is an important modulator of the synthesis, secretion, and concentration of serum lipoprotein. Conflicting reports have appeared on effect of dietary cholesterol on HDL levels.

T.A. Borden et al. in 1964 reported enhanced levels of HDL-c in rats fed cholesterol while D.E. Haft et al., 1962 and D. Kritchevsky in 1965 reported no change in HDL levels in cholesterol fed rats.

R. Reiser et al., 1966 and A.N. Howard et al., 1968 reported decreased level of HDL cholesterol in rats fed with high cholesterol diet.

K.A. Narayan 1971 demonstrated that HDL_2 decreased drastically about 50% in rats fed with high cholesterol diet. These results confirmed the earlier observation of Reiser et al., 1966 that rat serum HDL level was decreased irrespective of whether a saturated or unsaturated fat was used in the diet supplemented with cholesterol. In short term feeding studies, marked reduced in dietary fat and isocalorie increase in carbohydrate resulted in decrease in HDL cholesterol in conjuction with elevation of serum triglyceride and VLDL. studies of HDL composition have shown a decrease in ratio of apolipoprotein A-I to A-II and a decrease in HDL cholesterol to protein ratio (Schonfeld et al., 1976) consistent with a selective decrease in HDL_2 species (Blum et al., 1977).

There is evidence that substitution of large quantities of poly-unsaturated fat for saturated fat in diet can result in lower levels of HDL lipids and proteins (Nichaman et al., 1967). An increase in the P:S fat ratio from 0.25:1 to 4:1 in food diet fed to four normal subjects for five weeks resulted in reduction of HDL cholesterol and apolipoproteins A-I

concentration of 33 and 21 percent respectively, with an associated reduction in HDL_2:HDL_3 ratio (Shepherd et al., 1978). Other studies have however reported either no change (Lewis 1978, Shore et al., 1981) or increase (Jackson and Glueck, 1980) in levels of HDL cholesterol with feding of diets enriched in Polyunsaturated fats. High dietary intake of cholesterol, in the form of three to six egg yolk per day, has been reported to produce increase in apolipoprotein E- containing HDL- sub species in human (Mahley et al., 1978). This effect was seen whether or not there was an increase in total plasma cholesterol. Despite the fact that HDL containing apolipoprotein E represented only a minor fraction to the total HDL, its presence was shown to account for an increase of 2.6 to 4 times the binding of HDL the binding of HDL to LDL receptors of fibroblasts as compared to pretreatment HDL (Mahley et al., 1981). But this was not observed in another study (Applebaum et al., 1979). Recently it has been reported that level of HDL cholesterol and serum apolipoprotein A-I, but not apolipoprotein E increased with the feeding of diets high in both cholesterol and saturated fat (Tan et al., 1974).

A final consideration in evaluating the effects of dietary variables on HDL is that, while levels of HDL cholesterol and plasma apolipoprotein A-I are similar after overnight fast and the nonfasting state, (Henderson L.O. et al., 1980), changes in levels and composition of HDL have been shown to occur acutely after meals containing fat. Cholesterol, Phospholipid, and C-apolipoprotein levels in HDL_2 increases, and cholesterol in HDL decreases (Havel R.J. 1973, Baggio G. et al., 1980) in conjuction with transfer of chylomicron lipids to HDL during the course of their catabolism. Recently it has been shown that HDL apolipoprotein A-I levels increased when fat was consumed in divided doses over a 10-hours period, but not when the same amount of fat was ingested as a single load (Kay R.M. et al., 1980).

LOW DENSITY LIPOPROTEIN-CHOLESTROL (LDL-c)

LDL-c is generated by the degradation and removal of triglyceride from very low density lipoprotein (VLDL) in the plasma, their density is in the range of 1.019-1.063 and they contain apoprotein B_{100}. More than 75 percent of the total cholesterol present in the plasma is in the form of LDL-c.

One function of LDL is to supply cholesterol to a variety of extrahepatic parenchymal cells, such as adrenal cortical cells, Lymphocytes, muscle cell, and renal cells. In 1977 Goldstein hypothesised the concept of LDL receptor. The presence of these receptors have been confirmed by many laboratories. LDL receptors are present on the cell surface of liver, adrenal cortical cell, Lymphocyte muscle cell and renal cells. LDL that binds to this receptor is taken up by receptor mediated endocytosis and digested by lysosome within the cells. The cholesterol esters of LDL are hydrolysed by a lysosomal cholesteryl esterase, and the liberated cholesterol is used both for membrane synthesis and as a percussor for steroid hormone synthesis. Liver uses the LDL-c for synthesis of bile acids and for generation of free cholesterol which is secreted into the bile.

In humans around 80 percent of LDL is removed from the plasma each day by the LDL recepter pathway the remainder is degraded by scavenger cell system in phagocytic cells in reticule endothelial system.

DIET INDUCIAL CHANGES IN LDL-s

Diet high in fat and cholesterol cause an elevation in LDL in most animals (Mahley R.W. 1978). The response in man varies, but in those subjects who have an elevation in plasma cholesterol, there is an elevation in plasma LDL levels. In 1979 Deborah-applebaum et al. demonstrated significant rise of LDL level in human volunteers after feeding 5000 mg of egg yolk cholesterol per day for 30 days.

Age related difference in rise of LDL was demonstrated by Arora R.C. and Gupta Gulab et al. in 1987. They found out that rise of total serum cholesterol after feeding high fat, high cholesterol breakfast for one week was much more pronounced in young (20-30 gm) volunteers with major portion of rise being contributed by increased HDL. Contrarery, in older age person the rise of total serum cholesterol was less marked with LDL-c contributing mainly in the increased levels.

M.F. Baudet et al. demonstrated that, there was significant fall in level of LDL in five volunteers 3 hours and 5 hours often taking butter diet. They attributed this fall due to defect in VLDL hydrolysis

by serum lipases and due to metabolic blocking in liver or adipose tissue.

In addition to this the diet induced LDL are larger than LDL from the same species on low fat - low cholesterol diet. In a study performed by Rudel and co-workers in 1979 on rhesus monkey showed that, high cholesterol diet induced LDL have molecular weight which are 1.5 fold larger than those of control LDL. Further more St. clair and Leight in 1978 have reported that the diet induced, large LDL are capable of stimulating cholesteryl esterification and accumulation in smooth muscle cells to a greater extent than are normal LDL.

An additional alteration in the LDL, induced by the high cholesterol diets involve the apoprotein constituents. In normal LDL, the B-apoprotein is the major detectable apoprotein moiety, however in several species the LDL contain a variable amount of the E apoprotein following cholesterol feeding (Mahley R.W. et al., 1977, Rudal L.L. et al., 1979).

CONTROL OF PLASMA CHOLESTEROL LEVEL BY LDL RECEPTORS

A decade of intense investigation has established a central role for lipoprotein receptors in regulating

plasma cholesterol traffic. Operationally, the LDL/LDL receptor system can be considered the primary transport mechanism for endogenous cholesterol. LDL are generated in the plasma by the degradation of intermediate density lipoprotein (IDL). Generated LDL is removed relatively slowly from plasma by binding to LDL receptors in the liver and extra hepatic tissues, (Kita T. et al., 1982). In rabbits, rats, and hamsters, more than half of the total LDL receptors are located in the liver. However the precise distribution of these receptors in man is unknown.

REGULATION OF HEPATIC LDL RECEPTOR

Hepatic LDL receptors are suppressed whenever the livers content of cholesterol increases or its demand for cholesterol is reduced. Thus receptor suppression occurs when a high cholesterol diet is consumed, (Hui, D.Y. et al., 1981) or when bile acids are infused (Angelin B. et al., 1983). Conversely, LDL receptors increases when hepatic cholesterol synthesis is blocked by drugs compactin or mevinolin (Goldstein et al., 1982, and Bilheimer D.W. et al., 1983), when bile acid binding resins are given (Shepered J. et al., 1980), or when an ileal by pass is created (Spengel F.A. et al., 1982). Fasting has also been

shown to supress LDL recepter in rabbits (Goldstein J.L. 1982). LDL receptors can be stimulated by thyroxine (Thompson G.R. 1981) and by pharmacologic doses of estrogen (Winder, E.E.T. 1980). Hepatic LDL receptors decline when rabbits are fed a diet composed only of sucrose and casein (Chao Y.S. et al., 1982). In dogs, hepatic receptors fall with ageing (Mahley R.W. et al., 1981).

All of the changes in recepter activity alter the rate of uptake of LDL by the liver and cause reciprocal changes in plasma LDL levels. Whenever hepatic LDL receptors are suppressed, the plasma LDL level rises, conversely, whenever these receptors are induced, the plasma LDL level falls.

FAMILIAL HYPERCHOLESTEROLEMIA AND LDL RECEPTOR

Familial hyper cholesterolemia is best defined clinically, genetically, and biochemically disorder characterised by (a) selective elevation in the plasma level of LDL (b) deposition of LDL derived cholesterol in abnormal sites in the body (c) inheritance as an autosomal dominant trait with a gene dosage effect. It occurs at a frequency of about 1 in 500 person.

The basic defect is reduced number of LDL receptors. In normal person about 45 percent of the plasma LDL pool is removed from the plasma daily by the receptors, wheres in familial hypercholesterolemia hetrozygotes this value is 25-30 percent and in homozygotes it is about 15 percent. This recepter deficiency results in accumulation of LDL into the plasma, leading to raised level and premature atherosclerosis.

TRIGLYCERIODES AND VERY LOW DENSITY LIPOPROTEIN (VLDL)

The level of serum triglyceride (STG) rises considerably after fat ingestion. Rise in the triglyceride level after fat ingestion has been reported after giving different amounts of the fat load and measuring the blood levels at different time interval (Nikkila and Kenttinen 1962, Demborough, 1963).

Angervall in 1963 has reported a significant correlation between fasting, 3½ hours valve and 7½ hours valve of serum triglyceride postprandially.

Clefsky et al., in 1976 noted a biphasic plasma triglyceride surge with an initial peak occuring 1 to 3 hours after feeding and a secondary peak after 4 to 7 hours. The primary peak was accounted by increase in chylomicron levels in more

then 98% cases, wheres secondary peak represented rise in very low density lipepretein (VLDL) level in 82%.

In 1957 Richard J. Havel concluded that increment in the concentration of triglycerides in the serum following ingestion of fat is entirly the result of an increase in their concentration in VLDL.

Excess production of VLDL and triglyceride is more often due to secondary abnormalities than to primary factors, perhaps the most common cause is high caloric intake associated with obesity, excess alcohol and excess carbohydrate. Increased levels are also found in diabetes mellitus, nephrotic syndrome and hypothyroidism with obesity. Delayed clearance of triglyceride from the serum is noted in cases of ischamic heart disease after high fat diet (Arora R.C. et al., 1967, David F., Brown et al., 1961).

VLDL → REMNANTS

Also known and Beta VLDL these particles are smaller than normal VLDL and contain more cholesterol. Both of these characteristics impart atherogenic potential to VLDL remnants.

BETA VLDL IN CHOLESTEROL FED MAN

In addition to a report by Mistry P. et al. in 1976 that Beta VLDL can be induced by cholesterol feeding in man, priliminary studies from the Gladstone foundation laboratories for cardiovascular disease indicate that certain individual respond to high fat, high cholesterol diet by producing lipoproteins which are capable of delivering cholesterol to macrophages. The Beta VLDL may occur transiently as minor components of the human plasma fractions after diets high in fat and cholesterol are consumed, and may cause repeated cholesterol deposition in cells in the arterial wall over the years. The Beta VLDL, either chylomicron remnants or hepatic lipoprotein may represent the atherogenic particle postulated several years ago by Zilversmit. This alteration in the lipoprotein fraction may represent the most significant diet induced changes in lipoprotein predisposing to accelerated atherosclerosis.

SERUM TRIGLYCERIDE AND EXCERCISE

Reduced level of triglycerides are found after exercise (Cohen & Goldberg, 1960). On the other hand, Billimoria et al., 1959 found that the alimentary lipemia occured early in exercising than in resting

subjects. The explanation put forward for decreased levels of triglyceride after exercise is that working muscles directly utilise triglycerides for energy production.

CHYLOMICRONS

Chylomicrons are large lipoprotein particle containing dietary triglyceride and cholesterol. They are vehicle of lipid transport in exogenous pathway. There chylomicrons are secreted into the intestinal lymph and pass into the general circulation for transport to the capillaries of adipose tissue and skeletal muscle where they are acted upon by lipoprotein lipase liberating free fatty acids and monoglyceride. The remaining particle deprived of triglyceride is termed as chylomicron remnant which is rich in cholesteryl ester. This remnant travels to the liver, where it is taken up by chylomicron remnant receptor and metabolised.

Different fats give rise to specific type of chylomicrons. Triolein gives large distinctive spherical chylomicrons while those seen after tristearin are creamy and vary in shape and are comparitively small.

Chylomicrons are rapidly cleared from the plasma and normally are not present after an overnight fast. The detection of these particles in fasting plasma is always abnormal and may indicate presence of other hyper lipidemias.

The simplest method to detect chylomicron in post prandial state is "creaming in the cold". Increased levels of chylomicron in the plasma may be found in cases of genetic defect involving the enzyre. Lipoprotein lipase and in familial from of hypertriglyceridemia.

ATHEROSCLEROSIS : A POST PRANDIAL PHENOMENON

The possibility of atherosclerosis being a post prandial phenomenon was first proposed by Zilversmit in 1973. He hypotesised that chylomicron remnant or Beta VLDL may occur transiently as minor components of the human plasma fractions after diet high in fat and cholesterol is consumed. And this may cause repeated cholesterol deposition in cells in the arterial wall over the years, while the fasting cholesterol level may remain normal during the life time.

If atheregenais is a post prandial phenomenon then premature CAD must be common in hyperchylomicronemic states. However in familial lipeprotein lipase deficiency enormous quantities of chylomicrons accumulate in plasma, but accelerated atherosclerosis has not been reported (Fredrickson D.S. et al., 1978).

ATHEROSCLEROSIS AND LIPID LIPOPROTEIN LEVELS

(1) Total serum cholesterol (STC)

Elevated STC is a risk factor for coronary heart disease. At the level of 220 mg/dl the incidence of coronary artery disease (CAD) is nearly two fold as compared to level of 180/dl (Kannel et al., 1971). Similarly patient with proved coronary heart (CAD) disease have significantly higher cholesterol concentration then patient without CAD (Cohn et al., 1977).

TRIGLYCERIDES

Several studies have shown that an elevation of plasma triglycerides is common in patients with CAD (Albrink et al., 1959, Hulley S.B. et al., 1980). Carlson and Bottiger in 1972 reported that rates of CAD rose linearly with increasing plasma triglycerides. However, there is currently great debate

as to whether VIDL is direct operative factor in producing CAD, or it is the association of increased LDL or decreased HDL level which are causative (Bilheimer 1972).

LDL CHOLESTEROL

LDL-c which constitutes about 75 percent of the total serum cholesterol is more specifically associated with CAD than is total cholesterol. It has been known for many years that the reduction of elevated LDL in other primate species is followed by regression of arteriosclerotic lesions in coronary arteries in large vessel (St clair 1983). We have now conclusive evidence in humans that reducing elevated LDL cholesterol will reduce the incidence of clinical events attributable to coronary arteriosclerosis (the lipid research clinics coronary primary prevention trial results, 1984).

HDL CHOLESTEROL

HDL level have an inverse relationship with coronary artery disease (Gordon et al., 1977). The ability of HDL cholesterol to predict the developing of coronary atherosclerosis has been estimated to be four times greater than LDL cholesterol and eitht times

greater than total cholesterol (Cordon et al., 1977). Each 10 ml/dl change in HDL cholesterol concentration is associated with 50% alteration in cardiovascular risk (Brensike, et al., 1984).

Sub classes of HDL can be fractioned by Zonal ultra centrifugation and include HDL_2 and HDL_3. Among these subgroup HDL_2 appears to have the strongest inverse relationship with CAD and accounts for different levels of HDL-c between men and women (Gofman et al., 1954). The possible Mechanism by which HDL cholesterol decreases atherosclerosis include.

1. Reversal of cholesterol transport from the peripheral cells to the liver for removal from the body (Miller and Miller 1975).

2. Inhibition of LDL cholesterol uptake by cells at the LDL receptor sites.

FAT TOLERANCE TEST AND ITS IMPLICATIONS

The concept of fat/cholesterol tolerance test is not entirely new. In 1907 neumann, after giving a fat load studied the quantitative lipid changes in form of chylomicron count after a fat load.

Introduction of isotopes, revolutionised the study of lipid metabolism. Brekowitz in 1963 pointed out that radio active fat tolerance is a better index for determining the functional state of lipid metabolism.

Zilversmit et al., 1979 brought forward the view that atherosclerosis may be a post prandial phenomenon with chylomicron and VLDL remnants of post prandial phase contributing to the development of atherosclerosis. This concept again aroused interest in determination of post prandial changes in lipid fraction often a meal rich in fat and cholesterol.

Subsequent work by Hanno Krauss et al., 1987 did not revealed any significant changes in serum total cholesterol often a heavy fat cholesterol load, but found significant difference in triglyceride levels.

Arora R.C. et al., in 1987 put forward the concept of triglyceride tolerence test which showed significant difference in peak levels of STG in normal healthy, patient of IHD and that of diabetes.

Diet prior to the loading test meal, may be decisive. Under metabolic ward conditions, significant difference in fat tolerence has been reported in healthy subjects on an isocaloric diet, when the

daily fat intake per kg of body weight was varied from 0.1 to 2 gm. (Harvel, 1957). The lowest intake gives the highest fat tolerance. In contrast to the above, no change in the fat tolerance has been noted when the fat content of the diet was raised from 40 to 54% for three weeks (Herlick, 1957).

In an interpopulation study, no difference has been reported in the fat tolerance of three different communities who consume 17,45 and 60% respectively of their total calories as fat (Beuchier and Bronte-Steward 1961).

Composition of the test meal also plays a important role. In human beings, glucose one hour and half an hour before as well as one and a half hour after a fat meal reduced or even eliminated the serum triglyceride rise (Albrink and Man 1956). Glucose addition to 131 I-labelled triolein caused a flatter triglyceride curve as compared to ingestion of the latter only (Berkowitz et al., 1959). The depression of free fatty acid (FFA) and serum triglyceride levels following increased glucose utilisation is thought to result from a decrease in the mobilisation of fatty acids from the fat depot of the body (Gordon 1957).

There is also evidence that an increase in hepatic fat synthesis may be important in the reduction of the serum FFA levels (Shoemaker et al., 1960).

Long term studies of the effect of dietary protein on lipid level indicate that low protein intake is accompnied by a depression of serum lipids (Olson et al., 1957).

In 1962 James F. Sullivan demonstrated that increasing the relative content of protein in a meal results in higher levels of serum triglycerides in the post prandial period.

In 1957 Richard J. Havel demonated fall in cholesterol level 4 hours after taking high fat diet in two male subjects.

FACTORS MODIFYING FAT TOLERANCE

(a) Age

Fat tolarance and age have shown differenee responses. Chylemicron count has been shown to rise more after a fat load in subjects more than 50 years as compared to the younger group (Becker et al., 1949, 50) similar results were found in turbidity measurements

(Marder et al., 1952, Schwartz et al., 1952). Using the same chylomicron counting principles, exactly opposite finding have been observed, and significantly lower chylomicron count in response to fat loading in older subjects, over 50 years as compared to younger subjects has been seen (Grunner and Hilden, 1953).

In a more illustrative work by Herzstein et al., 1953, it was observed that the total fats persisted longer in serum after fat loading in older subjects.

(b) Body weight

No significant correlation between body weight and the duration of lipemia in response to fat meal has been seen (Barritt, 1956). The fat tolerance rose appreciably after weight reduction was enforced.

(c) Exercise

It has been observed that at rest the lipid level of normal subjects increased by 42% after 3 hours of fat meal and the maximum was attained after 4 hours, while at work these figures were 34% at 3 hours (Nissen, 1931). Higher chylomicron counts

after fat loading in person at rest than in persons at work have been seen (Harder et al., 1952).

(d) Smoking

In habitual smoker, response to a fat meal indicated a lower post prandial rise in serum fat than to non smokers (Kenttinen and Rajasalmi 1963). One cigarette per hour caused the chylomicron count to rise in a group of young subjects but not in two elderly subjects (Harder et al., 1952).

REPRODUCIBILITY OF FAT TOLERANCE

By large, fat tolerance curve is reproducible over a period of six month with very little variation (Nortem, 1950, Osmon et al., 1957). However Bronte Stewart and Blackburn 1958 found considerable variability in response to the same fat load. Although those who exhibited a "high curve" continued to do so and vice versa.

HYPERLIPIDEMIA AND DIABETES

Hyperlipidemia is a relatively common problem in patients with poorly controlled diabites Mellitus associated with abnormal lipid metabolism, diabetics tend to have higher incidence of hypertension, obesity and 2-3 fold increase in cardio vascular morbidity

and mortality when compared with nondiabetics (Kannel W.B. et al., 1979). Many factors appear to contribute to this enhanced, atheromatous process in diabetes, including alteration in platlet function, clothing factors, arterial smooth muscle cell metabolism and possibly blood pressure regulation (Ganda, O.P. et al., 1980). Nevertheless, changes in plasma lipoprotein levels in diabetes remain of the most important associated risk factors in term of accelerated atherosclerosis (Santem, R.J. et al., 1972). In addition, diabetees may have altered lipoprotein structure and metabolism independent of increase in plasma lipid levels (Eckel R.H. et al., 1981, Howard, B.V. et al., 1978, Schonfeld, G. et al., 1974) and these altered lipoprotein may be associated with accelerated atherosclerosis. It is generally appreciated that anatomic evidence of accelerated atherosclerosis frequently develops in insulin dependent diabetic patient, 10-15 years after onset of diabetes.

ABNORMAL LIPOPROTEIN METABOLISM IN DIABETIC PATIENT

Hypertriglyceridemia is the most common lipid abnormality observed in diabetic patients. This is usually caused by accumulation of very low density

lipoprotein (VLDL) and rarely, chylomicrons in plasma (Nikkila, E.A., 1973). Hypercholesterolemia may also develop secondary to increased VLDL levels, though changes in plasma low density lipoprotein (LDL) and high density lipoprotein (HDL) levels also occur with variable degrees of diabetic control. Various mechanism have been reported to account for abnormal lipid metabolism, and this appear to depend on both the type of diabetes, and degree of insulin deficiency. Removal of triglyceride from plasma into adipose tissue requires two major processes.

(1) Hydrolysis of triglyceride to FFA catalyzed by lipoprotein lipase.

(2) Esterification of the fatty acids in the adipocyte with alpha-glycerophosphate derived from glucose.

Activity of lipoprotein lipase depends on amount of insulin present in the circulation. Its activity is inhibited by catecholamines, adrenocorticotrophic hormone, glucagon, and thyroid stimulating hormone (Robinson and Wing, 1970). Thus in insulin deficient diabetics activity of lipoprotein lipase is considerably decreased resulting into elevated

level of triglyceride.

GENETIC FACTORS OF CORONARY HEART DISEASE (CHD)

The genetic aspects of CHD have been extensively evaluated. Familial clustering of CHD strongly suggests that genetic factors play an important role in etiology (Deutscher et al., 1970, Epstein, 1964, Rose, 1960). Some studies suggest that familial aggregation of CHD may be influenced both by genetic characteristics of various risk factors and by common environmental conditions encountered by family members (Deutscher et al., 1970 : Epstein, 1964, Rissanen and Nikkila, 1977, Thomas, 1959, Thomas, 1956, Goldstein, 1973).

Thus, epstein estimated that almost two third of familial aggregation of CHD may be accounted for by familial trends in blood pressure and cholesterol levels. Similarly Rissanen and Nikkila observed that familial trends of CHD could in part be explained by familial elevated serum lipids, hypertension and diabetes Mellitus. Slack and Evans, 1966 pointed out that members of the same family tended to share common environmental conditions such as diet, smoking and sedentary habits Deutscher et al., suggested that numerous factors may play a role in familial CHD which are basically determined by genetic influences

and then altered by environment. Thus, the concept of multifactorial genetic and environmental interrelationship of risk factors can explain familial aggregation of CHD (Johnson et al., 1965, Epstein, 1964). Further evaluation of genetic aspects of CHD has been derived from twin studies. Cederlof et al.(1967) noted a concordance rate of CHD of 21.7 percent in monozygotic twins, compared to 6.1 percent dyzygotic twins. Similarly Verschver (1958) observed a concordance rate of 19% and 8.5% respectively in monozygotic and dyzygotic twins. However such finding as suggested by twin studies may be explained by genetic attributes of risk factors which may be influenced by common environmental experiences. Not only is heredity though to be the most important risk factor of cardiovascular disease but also it may be the easiest and least expensive way to detect in children. James Nora (1980) has reported that, making no additional assumptions about environmental and other genetic factors. The single highest risk factor of cardiovascular disease in children is first degree relative with myocardial infaction before age 55 years. This conclusion is supported by a study of a random sample of 320 pedigrees in Utah, demonstrating that 80% of the men with coronary occlusions under 55 years of age clustered in 16% of

families. Several other studies have reported that risk factors of cardiovascular disease show parent child clustering (Boulton, 1979; Gluch, 1978; Marrison et al., 1980). These studies suggest that one of the factors that may explain the relationship between premature heart disease in parents and offspring, may be elvated level of serum lipids.

LIPOPROTEINS : PATHOGENIC ROLE

Low density lipoproteins (LDL) and intermediate density lipoprotein(IDL) enter the arterial intima from plasma in man at rates directly related to their plasma concentration (Meithaus et al., 1977; Nicoll et al., 1981) and accumulate particularly in region already atheromatous. Endothelial injury greatly enhances this process. The cholesterol of atheromatous lesions is prinicpally derived from plasma (Zilversmit, 1968). The interactions or LDL with cells of atheromatous plaques have been studied in some detail. Smooth muscle cells and fibroblast have receptors that mediate uptake of LDL (Goldstein and Brown, 1974 and Bierman and Albers, 1979) its cholesterol is released by lysosomal degration. Macrophages lack these receptors but acquire lipoprotein cholesterol by other processes.

including receptor mediated uptake of altered LDL. In contact with cultured endothelial cell, LDL is modified, permitting macrophages to degrade it (Henriksen et al. 1981). Normally equilibrium is maintained in influx and efflux of cholesterol from the cell. Transport of cholesterol from peripheral cells to the liver may be mediated by HDL, soon after Miller and Miller 1975 advanced the concept that this function of HDL by favouring mobilisation of cholesterol from arterial wall, might explain the inverse relationship between HDL and risk of coronary heart disease.

THE LIPOPROTEINS : PREDICTORS OF CHD

The association of lipoproteins with coronary heart disease has been studied in depth in epidemiological studies. These associations are strong, predictive and independent of other risk factors.

Concentration of LDL cholesterol are directly related to and are predictive of the risk of coronary heart disease over a wide age range (Gordon et al. 1981). Mortality rates from coronary heart disease in different communities are directly and linearly related with serum concentrations of cholesterol and

LDL cholesterol (Lewis et al. 1978). HDL cholesterol concentrations are even more strongly predictive of the risk of coronary heart disease in most (Gordon et al., 1981; Goldbourt and Medalie, 1979) but not in all studies (Wiklund et al. 1980). The relation being inverse, but unlike LDL, HDL cholesterol concentration do not correlate inversely with mortality rates from coronary heart disease in different countries.

Hyperlipidemia usually runs in family, screening for hypercholesterolemia at age of 12 years, is fairly predictive of adult hypercholesterolemia close to 50% of the top quintile (88%) for cholesterol, were similarly placed at followup, nine years later, of interest was the observation that those who dropped out of the top quintile at followup had a lower incidence of obesity, smoked less and were more active (Orchard et al. 1983).

In childhood HDL contributes proportionately more to the total cholesterol concentration. In a survey of 6775 school children a substantial proportion of those with hypercholesterolemia were attributable to high HDL cholesterol levels (Morrison et al. 1979). The ratio of total cholesterol to HDL cholesterol

is about as efficient as any other lipid profile (Kannel et al. 1979). A ratio of 5 indicates the average high risk in affluent western populations, and ratio exceeding this are definite cause of concern within the range of serum cholesterol valve that are commonly encountered. A more optimal ratio is in the vicinity of 3.5 corresponding to half the standard risk and resembling that found in low CHD incidence countries (Gordon et al. 1982).

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MATERIAL AND METHODS

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The case material for the present study consisted of healthy male and female junior doctors and students of M.L.B. Medical College, plus patients, and their healthy attendents attending O.P.D. and wards of M.L.B. Medical College hospital Jhansi.

Informed consent was taken from every case, in each case a detailed history was elicited and a meticulous clinical examination and investigation were caried out, to groups, these cases into following categories:

Group A

It consisted of healthy male voluenters in the age group of 20-60 years. The number of subjects included in this group was 31.

Group B

Second group consisted of 20 healthy female volunteers in the age range of 20-60 years, most of them were junior doctors, students and staff nurses working M.L.B. Medical College, Jhansi.

Group C

Third group consisted of 11 young healthy volunteers both male and female who were first degree relatives of patients of ischaemic heart disease. The age range of this group was 20-30 years. In each case either mother or father had an ischaemic episode in the past.

Group D

In this group eitht patient of dibetics were included. 3 cases were of Juvenile onset diabetes and 5 cases were of maturity onset diabetes.

A detailed dietary history was elicited to assess the amount of fat consumed daily and weekly, by these subjects in their usual routine diet. Specific consideration was given to record the weekly amount of ghee and its type (saturated/ unsaturated) oil and its type, milk and milk products, eggs, and food additives. Majority of the subjects were hostelers eating a common type of food in hostel mess, thus per head consumtion of fat was calculated by giving consideration to the total amount of oil of ghee purchased monthly and number of members eating in the same mess.

Any recent change in diet, oral or parentral medication before and during the study were noted. Hospitalised patients were given the diet from the hospital for one week prior to the test.

DESIGN OF TEST

All the subjects were asked to have their dinner at 6.00 P.M. on the privious nitht and not to take anything except water till the next morning. Fasting blood samples were taken at 8.00 A.M. in the recumbent posture, without producing venous stasis (Keereelman et al.,1961). After this, they were given a test meal. Consisting of 3 boiled egg with 250 ml of boiled sweetened milk. This supplied around 750-800 mg of egg yolk cholesterol.

Post prandial blood samples were taken at 9.00 A.M. and 11.00 A.M. In some cases additional ample at 10.00 A.M. was also taken. During the test the subjects were watching T.V. film and were not allowed to take anything except water. Smoking was prohibited during the test period. Plasma was seperated from the blood samples and the following tests were performed.

(I) TOTAL SERUM CHOLESTEROL

Cholesterol estimation was done by one step method of wybenga and pileggi 1970 utilising commercial kits supplied by "SPAN" diagnostics.

(II) SERUM TRIGLYCERIDE ESTIMATION (STG)

Estimation of serum triglyceride was done by acetyle acetone method, using kit supplied by "ETHNOR".

(III) SERUM HDL - CHOLESTEROL ESTIMATION

This test was conducted by using commercial kit supplied by ETHNOR.

(IV) ESTIMATION OF VLDL - CHOLESTEROL

VLDL cholesterol was derived using formula given by Fried Wald et al. 1972.

$$\text{VLDL - Cholesterol} = \frac{\text{Serum triglyceride}}{5}$$

It is valid till STG value are less than 400 mg%.

(V) CALCULATION OF LDL - c

LDL - c was also estimated by using formula given by Fredrickson, D.S. 1972.

$$\text{LDL-c} = \text{Serum cholesterol} - \left(\frac{\text{STG}}{5} + \text{HDL-c} \right) \text{mg\%}.$$

Statistical analysis of the data was done by using paired 't' test and student 't' test.

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OBSERVATIONS

OBSERVATIONS

The present work was conducted on 31 male healthy volunteers 44% (Group A), 20 female healthy volunteers 28% (Group B), 11 male and female young healthy first degree relatives of patients of coronary artery disease 16% (Group C) and eitht patients of Diabetes 12% (Group D).

1. Group A :- It comprised of 31 healthy male volunteers in the age range of 20-60 years with mean age of 38.29±12.97 years, and mean weight of 61.67±8.75 kgs. Their other general particulars are depicted in table 1.

2. Group B :- It comprised of 20 healthy female volunteers in the age range of 20-60 years with mean age of 31-95±12.68 years and mean weight of 49.8±6.73 years. Their other general particulars are given in table 2.

3. Group C :- It comprised of 11 healthy male and female volunteers who were first degree relatives of patients of coronary artery disease. Their mean age was 25.09±2.02 years and mean weight 64.63±11.17

kgs. Their other general particulars are given in table 3.

4. Group D :- It consisted of total of eight patients of diabetes, out of which 3 were of juvinile onset diabetes and rest 5 of maturity onset diabetes, their mean age was 52±8.57 years and mean weight 58±7.48 kgs. Their other general particulars are given in table 4.

CHANGES IN SERUM TOTAL CHOLESTEROL

Healthy females (group B) had a higher fasting serum total cholesterol than males (group A). However the highest levels were observed in diabetic subjects (group D)Table 5). These difference were statistically insignificant (P 7.05). In all the groups, single high cholesterol test load resulted in fall in STC levels in first hour. The magnitude of fall was maximum in subjects of group A and minimum in first degree relatives of patients of coronary artery disease (Group C). Three hour STC valve in all the groups showed a rise over one hour valve but in remained below fating valve except for in group C where it surpassed the fasting valve. None of the feeding induced changes were statistically significant (Table 6).

Table 1

Showing general particulars of subjects of Group A

Characteristics	Number	Percent
Occupation		
1. Junior Doctors	13	42
2. Business man	04	12
3. Technician	02	07
4. Manual workers	09	29
5. Ward boys	03	10
6. Students	—	—
Physical Activity		
1. Sedentary	05	16
2. Moderate	23	74
3. Heavy	03	10
Dietary Habits		
1. Vegetarians	11	35
2. Non Vegetarians	20	65
Smoking		
1. Smoker ⩾10 cigrater/day	06	19
2. Nonsmoker or ⩽10/day	25	81
Fat consumption		
1. Low (⩽ 50gms/day)	13	41
2. High(⩾100gm/day)	05	18
3. Moderate(50-100gm/day)	13	41

Table 2

Show general particulars of subjects of Group B

Characteristics	Number	Percent
Occupation		
1. Students	04	20
2. Staff Nurses	07	35
3. House wives	45	25
4. Doctors	04	20
Physical Activity		
1. Sedantary	04	20
2. Moderate	16	80
3. Heavy	—	—
Dietary habits		
Vegetarian	07	35
Nonvegetarian	13	65
Fat consumption		
Low(/50 gms/day)	15	75
High(7100 gms/day)	05	25

Table 3

Showing general particulars of subjects of Group C

Characteristics	Number	Percent
Occupation		
- Doctors	08	12
- Students	02	18
- Staff nurse	01	10
Physical Activity		
- Sedentary	—	—
- Moderate	09	82
- Heavy	02	18
Dietary Habits		
- Vegetarians	03	27
- Non vegetarians	08	73
Smoking		
- Smokers 7 10/day	03	27
- Non smokers or ∠10/day	08	73
Fat Consumption		
- High (7 100 gms/day)	—	—
- Low (∠ 50 gms/day)	01	09
- Moderate (50-100 gms/day)	10	91

Table 4

Showing general particulars of subjects of Group D

Characteristics	Number	Percent
Occupation		
- Business man	01	12.5
- House wives	02	25.0
- Labourers	04	50.0
- Student	01	12.5
Physical Activity		
- Sedentary	04	50.0
- Moderate	04	50.0
- Heavy	—	—
Dietary Habits		
- Vegetarian	05	62.5
- Nonvegetarian	03	37.5
Smoking		
Smokers 7 10 cigrater/day	02	25.0
Nonsmoker or ⟨10 cigrater/day	06	75.0
Fat Consumption		
High (7100 gms/day)	—	—
Low (⟨50 gms/day)	06	75
Moderate (50-100 gms/day)	02	25

Table 5

Effect of high cholesterol test load on mean STC in Group A, B, C, D.

Group	Basal Fasting mg/dl I	1 hrs.after test load mg/dl II	3 hrs.after test load mg/dl III
A (n=31)	164.4±36.98	156.4±33.3	158.2±33.56
B (n=20)	185.2±36.2	179±39.38	183.2±41.4
C (n=11)	178.72±30.17	176.90±47.28	181.36±42.30
D (n=8)	206.87±64.75	197.87±45.68	205.5±39.26

CHANGES IN GROUP A:

The mean fasting STC of group A was 164.4±36.98 mg/dl (table 5). In the first hour after single high cholesterol test load, it showed a decline, however 3 hours after, there was a slight increase in STC levels, but it did not touched fasting basal levels. On statistical analysis, none of the changes were statistically significant (P7.05) when paired 't' test was applied (table 6).

Three types of responses were observed after giving single test load of high cholesterol. In some subject there was increase in STC level in the first hour after the test load. If this increase was more than 5 percent over the fasting value, this type of response was termed as A.I. In some subject there was a sharp fall in STC level after 1 hour. If this fall exceeded 5 percent of the fasting value, this was termed as A-III response. In the remainder cases, no effect was observed after administration of test meal. If the increase or decrease in 1 hours value was less than 5 percent of the basal, this was termed as A-II response.

On the basis of the above classification, there were 10 (32.25%) case of A-I type of response, whose fasting mean STC was 154.9±26.41 mg/dl and their 1 hour mean STC rose to 174.8±29.58 mg/dl (table-9). Difference in fasting and 1 hour value was highly significant ($P<0.005$). In A-II group there were 6 subjects (19.35%) in whom practically no change was observed 1 hour after test load. A-III type of response was most commonly observed. 15 subject (48.36%) whowed this type of response, their 1 hour value showed a decline from 170.56± 93.46 to 140.73±28.26 mg/dl. This difference was statistically significant ($P<0.005$)(table 9).

Table 9

Table showing three different type of responses in group A after administration of single high cholesterol test load, and their statistical significance.

Group	Fasting STC mg/dl	1 hr. mg/dl	't'	P
A I (n=10)	154.9±26.41	174.8±29.58	5.84	<0.005*
A II (n=6)	164.9±26.5	165.08±29.1	0.71	7.10
AIII (n=15)	170.56±43.4	140.72±28.2	3.95	<0.005*

* Denote 'P' value is highly significant by paired 't' test.

Effect of age

No clear cut correlation of fasting STC was observed with rising age. The highest level of STC was observed in the age group of 21-30 years. Then after STC level fell in age group of 31-40 years, slightly rose in age group of 41-50 years and again fell in age group of more than 50 years (Table 7). On giving single high cholesterol test load, all the groups showed a decling in STC levels in first hour. This feeding induced fall in STC levels was maximimum in young subjects of 21-30 years of age and diminished with rising age. After three hours STC levels started rising over one hour valve but were well below fasting valve except for that of group C. None of the feeding induced changes were statistically significant (P 7.05) (table 8).

Table 7

Effect of single high cholesterol test load on mean STC in various age subgroups of Group A

Age Group	I Fasting mean mg/dl	II After 1hr. mg/dl	III After 3hr. mg/dl
21-30yrs. (n=14)	179.6±44.18	165.4±35.17	169.64±37.09
31-40yrs. (n=7)	148.2±26.3	147.37±44.91	149.82±40.88
41-50yrs. (n=5)	157.9±23.7	151.0±19.02	149.4±20.4
7>50yrs. (n=5)	151.0±27.36	149.2±20.12	148.0±15.14

Those subjects who consumed a high fat diet had a higher fasting STC level as compared to those who consumed moderate or low fat. This difference was statistically significant (P<.005) (table 12). There was also difference in feeding behavior, subject who had mhigh consumption of fat daily showed a rise of STC level in 3 hrs. Value, while subject who consumed moderate to less fat showed a continuous decline in 1 hour and 3 hour value.

Table 12

Showing basal STC level of high, moderate and low fat consumers and their feeding behaviour in group A

Group	Fasting mg/dl	1 hrs. mg/dl	3 hrs. mg/dl
High n=5	217.4±42.22	182.4±36.23	197.6±40.16
Moderate n=13	198.3*±22.17	155.9±29.37	152.46±23.81
Low n=13	150.07*±27.39	146.92±33.08	148.87±30.58

* Denotes difference in level is statistically highly significant when compared to high fat intake group on applying student 't' test (P<.005).

EFFECT OF PHYSICAL ACTIVITY

5 subjects (10%) were doing heavy physical work daily, while 23 subjects 74% were doing moderate physical activity and remaining 5 subjects (16%) were sedentary.

Subjects who did heavy physical work, had a lower basal STC level as compared to moderate and sedentary subjects, this difference was statistically insignificant.

Table 14

Showing relationship between physical activity and STC and their feeding behaviour in group A

Group	Basal mg/dl	1 hr. mg/dl	3hrs. mg/dl
Heavy n=5	145.3±32.53	133.6±44.54**	153.6±35.90**
Moderate n=23	169.7±38.87*	160.95±33.98**	161.06±36.67**
Seentary n=5	151.0±27.3*	149.2±20.12**	148.0±15.14**

* Denotes difference is insignificant when compared with heavy physical activity group by student 't' test.

** Denotes difference is insignificant when compared with respective fasting value by paired 't' test.

In all the three groups 1 hour after giving high cholesterol test load, a fall in STC valve was observed.

Sedentary subject showed very little change of STC on feeding, while in other two groups, the fall in STC in 1 hr. after feeding was of greater magnitude, although it was statistically insignificant. In heavy physical activity group, 3 hour STC value rose and crossed the fasting value.

CHANGES IN STC IN GROUP B

The mean fasting STC of group B was 185.2±36.2 mg/dl (table 5). The level of STC fell after 1 hour after feeding and in 3 hours showed some rise, but still the 3 hour value was less than the fasting value. All the changes were statistically insignificant on applying paired t test (table 6).

Again three different types of behaviour were observed 1 hour after feeding. In 5 case (25%) there was a rise of STC by more than 5% of the basal value, in 6 cases (30%) no change was observed 1 hour after feeding, and in 9 cases (45%) STC fell by more than 5%, 1 hour after feeding. There subgroups were labelled as B_I, B_{II} and B_{III} respectively. The change of STC in group B_I and B_{III} was highly significant (table 15).

Table 15

Showing three different type of responses after administration of single high cholesterol test load and their statistical significance in groupB

Sub group	Fasting mg/dl	1 hr.after mg/dl	't'	'p'
B_I (n=5)	187±26.34	200±25.1	9.02	<.001*
B_{II} (n=6)	189±46.29	187±48.47	0.71	<.01
B_{III}	181.6±37.4	163.1±35.5	7.096	<.001*

* Denotes 'P' value is highly significant by paired 't' test.

EFFECT OF AGE ON STC

Rising levels of fasting basal STC has observed with the increase in age up to 50 years (table 16) after that in 50-60 years sub group, the fasting level actually fell.

The difference in fasting value was found to be statistically significant in 31-40 years and 41-50 years group when compared to 21-30 years sub group (table 16).

On giving high cholesterol test load, young subjects in the age range of 21-30 years showed a fall in STC value 1 hour after and this fall continued till 3 hours. This fall in 1 and 3rd hour was found

to be significant statistically (table 17). In all the remaining age groups (31-40 years, 41-50 years and 750 years) the level of STC actually started rising 1 hour after test load and continued to rise even after 3 hours. However the magnitude of rise was maximum in subject of more than 50 years of age (table 16). Rise of this group was statistically significant and in the rest rise was insignificant (table 17).

Table 16

Effect of single high cholesterol test load on mean STC in various age sub groups of group B

Age groups	I Basal Fasting mg/dl	II After 1 hr. mg/dl	III After 3 hrs. mg/dl
21-30yrs. (n=12)	170.3±30.1	159±31.5	158.4±28.5
31-40yrs. (n=6)	212.3±38.8*	214.6±38.27	235±22.5
41-50yrs. (n=3)	224.3±21.23**	225±7.07	226±20.6
750 yrs. (n=2)	175±21.0	185±14.5	190.5±13.5

* Difference in significant statistically when compared with 21-30 yrs. group by student t test.

** Difference is highly significant statistically when compared with (21-30yrs) by student t test.

Table 17

't' and 'P' value of difference in STC (intragroup) comparison based on different age groups in group B

Groups		t	P	Degree of freedom
21-30yrs. (n=12)	I : II	2.96	<.05*	11
	I : III	2.59	<.05*	11
	II : III	0.11	>.10	11
31-40yrs.	I : II	0.39	>.10	02
	I : III	1.55	>.05	02
	II : III	1.79	>.05	02
41-50yrs. (n=3)	I : II	0.06	>.1	02
	I : III	0.37	>.10	02
	II : III	0.10	>.10	02
750yrs. (n=2)	I : II	1.61	>.05	01
	I : III	2.06	<.05*	01
	II : III	5.0	<.005**	01

* Denotes 'P' value is statistically significant (P<.05).

** Denotes 'P' value is highly significant (≤ P<.005).

EFFECT OF FAT CONSUMPTION ON STC

5 subjects 25% consumed high fat (7100 gm) daily while rest 15 subject 75% consumed low fat daily.

Subjects with high daily fat consumtion had a much higher fasting serum total cholesterol, then subjects with low fat consumtion. This difference in level was statistically significant (table 18). High fat consumers had very little effect of high cholesterol test load in 1 hour, and then subsequently in 3 hours they showed a steady rise in STC crossing fasting value (table 18).

Table 18

Showing difference in STC of high and low fat consumers and their feeding behavior of group B

Group	Fasting mg/dl	1 hr. after mg/dl	3 hrs. after mg/dl
High fat in take (n=5)	220±32.21	219.2**±27.91	226.4**±29.80
Low fat in take (n=15)	173.6±30.08	166.8**±33.87	168.8**±34.4

t = 2.98*

P = ∠.01

* Denotes difference is significant between fasting STC of two group by student 't' test.

** Denotes difference in insignificant when compared with respective fasting value by paired 't' test.

Low fat consumers, showed a decline of STC in 1 hour and subsequently small rise of 2 mg% in 3 hours. All the feeding induced changes were of insignificant value (table 19).

EFFECT OF PHYSICAL ACTIVITY ON STC

4 subjects (20%) were sedentary and remaining 16 (80%) were doing moderate physical activity daily.

Sedentary subjects had a much higher fasting serum total cholesterol level, as compared to subjects doing moderate physical activity. However the difference was statistically in significant (P>.05)(table 20)

Table 20

Showing effect of physical activity on basal STC and their feeding behaviour in group B

Group	Fasting mg/dl	1hr.after mg/dl	3 hrs.after mg/dl
Sedentary (n=4)	202.5±41.06	210.2±33.2**	219.2±34.9**
Moderate (n=16)	180.8±34.9*	172.3±37.9**	175.6±39.2**

* Denotes insignificant difference between fasting value of two groups by student 't' test.

** Denotes insignificant difference when compared with respective fasting value by paired 't' test.

3- CHANGES OF STC IN GROUP C (First degree relatives of patients of CAD)

There were 3 female and 9 male subjects in this group. The mean fasting STC was 178.72 30.17 mg/dl (Table 5), this level fell very little 1 hour after test load, and again rose in 3 hours crossing fasting valve. All of these changes were statistically insignificant (P>.10). Six out of eleven subjects (54.5%) showed a rising trend of STC 1 hour after test load, one subject showed no change in STC valve, while remaining four subjects showed a decline in STC after feeding.

EFFECT OF FAT CONSUMTION

10 subjects (91%) consumed moderate amount of fat daily while only 1 subject (9%) had a low daily intake of fat.

Basal STC levels was higher in subjects with moderate fat consumtion than with low fat consumer group. This difference was statistically not significant (P>.05) Table 23.

Moderate fat consumer showed slight fall in STC 1 hour after feeding and then again rise in STC level after 3 hour, while in low fat consumer STC level initially rose in 1 hour followed by a fall in third hour. The change was statistically insignificant in both the cases.

Table 23

Table showing difference in STC in moderate and low fat consumer, and effect of feeding high cholesterol load in group C

Group	Fasting mg/dl	1 hour after mg/dl	3 hours after mg/dl
Moderate (n=10)	179.4±31.7	174.9**±49.34	182.9**±42.9
Low (n=1)	172.0	197.0**	166.0**

$t = .70$

$P = 70.10^{*}$

* Denotes 'P' valve is not significant when fasting valve of both groups are compared by student 't' test.

** Denotes 'P' valve is not significant when compared with preceding valve by paired 't' test.

EFFECT OF PHYSICAL ACTIVITY

Nine subjects (81%) were doing moderate physical activity daily while two subjects (19%) were heavy workers. The fasting STC level was higher in subjects doing moderate physical activity than with those doing heavy labour. However the difference in level was statistically insignificant ($P7.05$)(Table 24). Subjects of both the subgroups

also showed different behaviour on feeding. In moderate physical activity groups STC rose progressively from fasting to 1 hour and 3 hour, while in heavey physical activity group STC levels intitially fell in first hour and then slightly rose in third hour. The change in both the groups was statistically insignificant (P>.05) (Table 24).

Table 24

Table showing basal STC level in moderate and heavy physical activity groups and effect of feeding single high cholesteral test load in group C subjects

	Fasting mg/dl	1hour after feeding mg/dl	3hours after feeding mg/dl
Moderate (n=9)	183.3±30.89	185.0**±49.6	188.2**±41.7
Heavy (n=2)	158.5±19.09	145.5**±14.8	150.5**±3.5
	t = 1.9		
	P = 7.05*		

* Denotes P valve is insignificant by student 't' test

** Denotes P valve is insignificant when compared with fasting valve by paired 't' test.

CHANGES IN STC IN GROUP D

Patients of type I or Juvinile diabetes had a much lower based STC level as compared to patients of type II or maturity onset diabetes. The difference was statistically highly significant (P<.001)(Table 26). On feeding single high cholesterol test load in these

group, two distinct behaviors were observed. In type I diabetics STC levels started rising from one hour and rise continued till three hour. Three hours rise was statistically highly significant (P = <.001). On the contrary feeding in type II diabetics resulted in fall in STC level in first hour and it continued into third hour. None of the change was statistically significant (Table 26).

Table 26

Table showing basal STC level in type I and type II diabetic subjects of group D and effect of feeding high cholesterol test load in them

Group	Basal fasting mg/dl	1 hr. after feeding mg/dl	3 hrs. after feeding mg/dl
Type I (n=3)	138.3±12.4	148.3±12.4	171.0±15.7**
Type II (n=5)	248±35.5	227.6±24.3	226.2±29.43
	t = 5.09		
	P = <.001*		

* Denotes highly significant P (P<.001) when compared by student 't' test.

** Denotes highly significant P (P<.001) when compared with fasting and preceeding valve by paired 't' test.

EFFECT OF FAT CONSUMPTION

6 subjects (75%) were low fat consumers while 2 subjects (25%) were moderate fat consumers. Moderate fat consumers had significantly higher level of basal STC as compared to low fat consumers ($P < .05$). On giving single high cholesterol test load, low fat consumers showed a progressive rise of STC level in one and three hour, though this rise was statistically insignificant. On the contrary moderate fat in takers showed a progressive fall in their STC levels after feeding. Three hour fall was statistically significant ($P < .05$) (Table 27).

Table 27

Table showing basal STC level in low and moderate fat consumers subjects of group D and effect on STC on feeding single high cholesterol test load

Groups	Basal fasting mg/dl	1hr. after feeding mg/dl	3hrs. after feeding mg/dl
Low (n=6)	180.83±48.82	182.5±39.46	194.66±33.72
Moderate (n=2)	285±33.94	244.0±33.94	202.0**±12.0
	t = 2.75		
	P = <.05*		

* Denotes significant 'P' valve when compared by student 't' test.

** Denoted significant 'P' valve when compared with corresponding fasting valve by paired 't' test.

CHANGES IN HIGH DENSITY LIPOPROTEIN (HDL)

HDL cholesterol showed very little change on feeding single high cholesterol test load. Highest mean levels were observed in group B (healthy females) and lowest in group C. Only in group B slight rise of HDL-C was observed 1 hour after feeding. In rest of all the groups there was slight fall in HDL-C level 1 hour after feeding (Table 29).

The changes in valve of HDL-C after feeding was statistically in significant in all groups except in group C where 1 hour valve and 3 hour valve showed a statisical signicifant ($P<.05$) difference and in group D where the difference between fasting and 3 hour valve was statistically significant (Table 29).

Table 29

Effect of high cholesterol test load on mean HDL-C levels in group A, B, C and D.

Group	Basal fasting mg/dl I	1hr. after test load mg/dl II	3hrs. after test load mg/dl III
A (n=31)	46.07±6.34	45.9±8.02	45.71±6.99
B (n=20)	47.2±15.0	48.2±13.6	49.5±13.2
C (n=11)	43.5±11.5	42.2±10.80	46.1±11.5*
D (n=8)	47.1±8.27	46.7±7.7	45.1±10.4*

* Denotes significant 'P' valve ($P<0.05$) when compared with preceeding valve.

** Denotes significant 'P' when ($P<0.05$) compared with corresponding fasting valve.

I-Changes in Group A (Healthy males)

Fasting HDL levels was 46.07±6.34 which slightly fell in first and third hour after feeding.

EFFECT OF AGE

Highest levels of HDL-C was observed in age group of 41-50 yrs. No real correlation was observed between HDL-C level and age (Table 30). In group 21-30 yrs. and 31-40 yrs., there was a slight fall in HDL-C levels in first hour after feeding, but in remaining two subgroups (41-50yrs., and 750 yrs.) first hour valve showed slight rise over fasting valve. In all the cases the changes were statistically insignificant ($P<0.05$).

Table 30

Effect of single high cholesterol test load on mean HDL-C in various age subgroup of group A

Age group	I Fasting Basal mg/dl	II 1hrs. after mg/dl	III 3hrs. after mg/dl
21-30yrs. (n=14)	45.6±4.97	44.6±6.18	44.4±4.99
31-40yrs. (n=7)	44.3±7.05	43.5±8.8	43.21±6.78
41-50yrs. (n=5)	49.25±9.58	51.2±12.2	46.9±10.9
750 yrs. (n=5)	46.3±5.95	47.3±5.82	47.1±9.07

EFFECT OF SMOKING

There were 25 nonsmokers (80%) and 6 smokers subject (20%). Nonsmokers had a slightly higher level of HDL cholesterol than smokers, (Table 31) though the difference was statistically not significant. On feeding, non smokers showed a slight rise in HDL level in first hour followed by a fall in third hour. Contrary smokers on feeding showed a fall in HDL level in first hour followed by rise in third. Feeding induced changes in both the groups were statistically insignificant (P=/.05) (Table 31).

Table 31

Table showing based HDL level in smokers and non smokers and changes induced after feeding single high cholesterol test, load.

Group	Fasting Basal mg/dl	1hr.after feeding mg/dl	3 hrs. after feeding mg/dl
Nonsmokers (n=25)	46.11±6.83	46.42±8.3**	45.7±7.66**
Smokers (n=6)	45.78±5.93	43.69±5.63**	45.9±3.47**
	t = 0.3		
	P = 7.10*		

* Denotes insignificant P(P>.10) valve when compared by student 't' test.

** Denotes insignificant 'P' valve when compared with corresponding fasting valve by paired 't' test.

EFFECT OF PHYSICAL ACTIVITY

23 subjects (74%) did moderate physical activity, 5 subjects (16%) were sedentary and 3 subjects (10%) were doing heavy physical labour. Subjects who were sedentary or doing moderate physical work daily, had a higher level of HDL-C as compared to subjects doing heavy work. However the difference was statistically insignificant ($P>.05$). On feeding single high cholesterol test load no change in HDL-C levels were observed in moderate and heavy workers in first hour, but sedentary workers showed slight rise, after three hours rise of HDL-C was observed in heavy workers, fall in moderate workers and no change in sedentary. None of the changes observed were statistically significant (Table 32).

Table 32

Table showing basal levels of HDL-C and effect of feeding single high cholesterol test load in sedentary moderate and heavy workers subject of group A

Group	Basal fasting (mg/dl)	1hr.after feeding (mg/dl)	3 hrs.after feeding (mg/dl)
Sedentary (n=5)	46.3±5.95	47.3±5.82**	47.1±9.07**
Moderate (n=23)	46.87±6.38	46.49±8.62**	45.98±6.94**
Heavy (n=3)	39.16±1.75	39.0±1.0**	41.33±2.30**

** Denotes P valve is insignificant ($P>.05$) when compared with corresponding fasting valve.

EFFECT OF FAT CONSUMTION

13 subjects (42%) consumed low amount of fat same number consumed in moderate amounts and 5 subjects (16%) consumed high amount of fat daily.

Basal HDL levels were highest in subjects consuming low amount of fat (Table 34). This difference was statistically insignificant. On feeding single high cholesterol test load, HDL-C level rose in low fat takers while it decrease slightly in high and moderate fat consumer after 3 hour. After it 3 hours HDL-C levels rose in both high and moderate fat takers, while it fell in low fat consumers. None of the change observed was statistically significant (table 34)

Table 34

Table showing basal level of HDL-C in high, moderate and low fat consumers and their behaviour after feeding single high cholesterol test load

Group	Basal fasting mg/dl	1 hr. after feeding mg/dl	3 hrs. after feeding mg/dl
High (n=5)	46.74±5.80	43.9±5.93*	44.9±2.96*
Moderate (n=13)	44.0±5.05	43.8±5.38*	45.05±5.5*
Low (n=13)	47.87±7.41	48.76±10.25*	46.73±8.79*

* Denotes 'P' valve is insignificant when compared preceeding valve by paired t test.

CHANGES IN HDL IN GROUP B (HEALTHY FEMALES)

Basal mean HDL level in group B was 47.2 it rose progressively 1 and 3 hour after giving single high cholesterol test load. This change was statistically insignificant (table 29).

EFFECT OF AGE

Maximum HDL-C concentration in fasting state was observed in age group of 31-40 years and minimum in subjects of more than 50 years of age (table 36). All of the age group showed rise of HDL-C in first hour after feeding single high cholesterol test load. This rise continued in age groups of 21-30 years and 31-40 years in third hour also but in rest of the groups there was fall in third hour. All the feeding induced changes were insignificant statistically except, the third hour fall observed in 41-50 years of age sub group (table 36).

Table 36

Effect of single high cholesterol test load on mean HDL-C in various age subgroups of group B.

Age group	I Basal (mg/dl)	II 1 hr.after (mg/dl)	III 3 hrs.after (mg/dl)
21-30yrs. (n=12)	47.64±18.05	51.06±19.23	52.0±11.7
31-40yrs. (n=3)	55.3±12.5	56.5±9.96	63-5±1.32
41-50yrs. (n=3)	49.25±9.58	51.5±12.2	46.9±10.92
750yrs. (n=2)	46.5±5.95	47.3±5.82	47.1±9.07

EFFECT OF FAT CONSUMPTION

15 subjects (75%) consumed low fat daily while 5 subjects (25%) were consuming high fat daily. Fasting HDL-C levels were higher in low fat consumer group. This difference was statistically insignificant. On feeding single high cholesterol test load both the sub groups showed rise in first and third hours. Rise was much more marked in high fat consumer group. All the changes observed were statistically insignificant (Table 37).

Table 37

Table showing basal HDL-C level in low and high fat consumers of group B and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1 hr. after feeding mg/dl	3 hrs. after feeding mg/dl
Low (n=15)	45.56±15.98	46.82±13.70*	47.7±12.63*
High (n=5)	42.24±11.54	53.5±15.41*	55.2±11.9*

* Denotes insignificant P valve (P>.05) when compared with corresponding fasting valve.

EFFECT OF PHYSICAL ACTIVITY

16 subjects (80%) were doing moderate physical activity daily while 4 subjects (20%) were sedentary. Sedentary. Sedentary subjects had a higher HDL-C levels. This difference was statistically insignificant (Table 38).

On feeding single high cholesterol test load sedentary subjects showed a fall in HDL-C level in first and third hour, while in moderate physical activity group a rise in HDL-C levels were observed in first and third hour. All the changes observed were statistically insignificant (Table 38).

Table 38

Table showing basal HDL-C level in sedentary and moderately active subjects of group B and their behaviour on feeding.

Group	Basal fasting mg/dl	1 hr. after feeding mg/dl	3 hrs. after feeding mg/dl
Sedentary (n=4)	51.62±12.6	49.37±15.14*	48.0±16.56*
Moderate (n=16)	46.13±15.71	48.26±14.26*	49.96±11.74*

* Denotes insignificant 'P' valve when compared with fasting valve.

CHANGES IN HDL-C IN GROUP C (FIRST DEGREE RELATIVES OF PATIENTS OF CAD)

Fasting basal HDL-c level in this group was 43.5±11.5 mg/dl and on feeding levels initially fell in first hour, followed by rise in third hour. This rise was statistically significant ($P<.05$) table 29.

EFFECT OF SMOKING

Only 3 subjects (27%) were smokers and rest 8 subjects (73%) were non smokers. Smokers had a considerabely low basal mean HDL level as compared to non smokers. The difference however was statistically not significant ($P>.05$) (table 40). On feeding single high cholesterol test load, both groups showed fall in HDL-c levels in first hour, followed by rise in third hor. In smokers fall and rise of first and third hour was statistically significant (table 9).

Table 40

Table showing basal HDL-c level in smokers and non smokers subjects of group C and effect of feeding single high cholesterol load in them.

Group	Basal fasting mg/dl	1 hr. after feeding mg/dl	3hrs. after feeding mg/dl
Smokers (n=5)	36.03±4.7	33.26±9.98*	41.8±5.86*
Non-smokers (n=8)	46.35±12.2	45.55±9.55	47.8±11.8

t = 1.39

P = 7.05**

* Denotes significant P (P<.05), when compared with preceeding valve by paired 't' test.

** Denotes insignificant P valve when compared by student t.

EFFECT OF FAT CONSUMPTION

10 subjects (91%) consumed fat in moderate amounts while 1 subject 9% had a low daily consumtion of fat. Subjects with moderate fat consumtion showed a higher level of basal HDL as compared to subjects with low fat consumtion. On feeding single high cholesterol test load, low fat consumers showed a progressive fall in HDL-c level in first and third

hour, while moderate fat consumers had a intial fall of HDL level in first hour followed by rise in third. This rise was statistically significant, ($P<.05$)(table 41).

Table 41

Table showing basal HDL level in moderate and low fat consumers of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1 hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=10)	43.99±12.02	42.74±11.24	*47.14±11.01
Low (n=1)	39.0±0	37.0±0	36.0±0

* Denotes significant 'P' valve ($P<.05$) when compared t with preceeding P valve by paired t test.

EFFECT OF PHYSICAL ACTIVITY

9 subjects (82%) were doing moderate physical activity daily while remaining 2 subjects (18%) were doing high physical activity daily.HDL-c levels where higher in subjects doing heavy physical activity daily, though the difference was not statistically significant (Table 43). On feeding single high cholesterol test load, both the groups showed fall in HDL-c level

in first hour and rise in third hour. Rise in moderate activity group was statistically significant (p<.05) (Table 43).

Table 43

Table showing basal HDL-c level in moderate and heavy physical activity doing subjects of group C and effect of feeding single high cholesterol test load in them.

Group	Fasting basal mg/dl	1 hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=9)	43.34±12.55	41.86±11.41	48.5±11.8
High (n=2)	44.4±7.91	43.72±10.92	44.4±5.6

* Denotes significant 'PS (P<.05) when compared with preceeding valve by paired t test.

CHANGES OF HDL-C IN GROUP D (DIABETICS)

Diabetic subjects had the highest basal HDL-c level among all the group. In this group too type II diabetics had a higher basal HDL level as compared to type I diabetics (Table 44).

Table 44

Table showing basal HDL-c level in type I and type II diabetic subjects and effect of feeding single high cholesterol test load in them.

Groups	Basal fasting mg/dl	1 hr. after feeding mg/dl	3hrs. feeding mg/dl
Type I (n=3)	44.66±8.49	41.53±4.49	40.0±11.79**
Type II (n=5)	48-57±6-84	49.85±6-8	45.02±9-42

* Denotes significant P value ($P < .05$) when compared with preceeding value.

** Denotes highly significant P value ($P < .005$) when compared with fasting value.

On feeding single high cholesterol test load the group has a whole showed when a fall in HDL-c level 1 and 3 hours after feeding. Three hours fall was statistically significant when compared with fasting value (table 29). On subgroup analysis, type I diabetic subjects exhibited same feeding induced changes, i.e. fall in HDL-c level 1 and 3 hours after feeding, which was statistically highly significant ($P < .005$) (table 44). But type II diabetics showed

a rise in HDL-c level in first hour, followed by fall in third hour. Both the changes were statistically significant (table 44).

EFFECT OF FAT CONSUMPTION

6 subjects (75%) were low fat consumers, while remaing 2 subjects (25%) consumed moderate amount of fat daily. Moderate fat consumers had a high basal HDL-c level as compared to low fat consumers. The difference was statistically significant ($p<.05$) (table 45).

On feeding single high cholesterol test load in them, moderate fat intakers showed a rise in HDL-c level in first hour, followed by fall in third hour, but low fat intakers showed a fall in HDL-c level in first and third hour. All the changes were statistically sinsignificant (table 45).

Table 45

Table showing basal HDL-c level in low and moderate fat intakers in diabetic subjects, and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr. after feeding mg/dl	3hrs. after feeding mg/dl
Low (n=6)	43.83±6.7	43.0±4.2	39.75±8.0
Moderate (n=2)	96.87±0.17	97.87±1.59	94.5±4.5

Denotes significant P valve (P<.05) when compared with fasting valve of low fat intakes by student t test.

EFFECT OF SMOKING

6 subjects (75%) were nonsmokers, while remaing 2 (25%) were smokers. Smokers had a low basal HDL-c levels as compared to nonsmokers, but the difference was statistically insignificant (table 46).

On feeding single high cholesterol test load in them both the group showed a fall in HDL-c level 3 hours after feeding. None of the change was statistically significant (table 46).

Table 46

Table showing basal HDL-c valve in smokers and nonsmoker diabetic subjects, and effect of single high cholesterol test level in them.

Groups	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Nonsmokers (n=6)	47.64±9.99	47.04±9.13	43.4±11.30
Smokers (n=2)	45.5±3.53	49.75±1.76	42.3±0.7

CHANGES IN SERUM TRIGLYCERIDES (STG)

Diabetics had the highest basal fasting serum triglycerides levels, while healthy male volunteers had the lowest value.

On feeding single high cholesterol test load rise in STG was observed in all the groups after 3 hours.

The magnitude of rise was maximum in diabetic subjects and minimum in relative of CAD, where actually slight fall in STG levels was observed in first hour, followed by very little rise in third hour (table 48).

Table 48

Effect of high cholesterol test load on mean STG levels in group A, B, C, D.

Group	Basal mg/dl I	1hr.after mg/dl II	3hrs.after mg/dl III
A (n=31)	146.0±38.7	157.51±44.53	168.48±43.3
B (n=20)	189.54±56.35	193.61±64.54	213.3±69.94
C (n=11)	183.8±90.93	181.8±52.59	186.54±63.69
D (n=8)	204.37±62.7	226.8±99.79	236.6±65.14

* Denotes significant 'P' valve (P<.05) when compared with fasting valve.

** Denotes significant 'P' valve (P<.05) when compared with preceeding and fasting valve.

CHANGES IN GROUP A (HEALTHY MALE VOLUNTEERS)

This group has a basal STG of 146.0±38.7 mg/dl it showed a significant rise one and three hour after test load (Table 48).

EFFECT OF AGE

Basal STG level rose with increasing age till 30 years then after in subjects of 730 years it showed a slight decline. (Table 49).

On feeding single high cholesterol test load, rise in STG levels were observed in all the age groups, but the magnitude of rise was maximum in younger age group and diminshed with increasing age. Only in 21-30 years age group. The rise was significant one and three hours after feeding (Table 49).

Table 49

Effect of single high cholesterol test load on mean STG in various subgroups of Group A.

Age group	I Basal valve mg/dl	II 1 hr.after mg/dl	III 3 hrs.after mg/dl
21-30yrs. (n=14)	124.92±32.85	134.5±33.76	150.2±28.9
31-40yrs. (n=07)	146.57±32.6	170.57±55.01	167.4±60.45
41-50yrs. (n=5)	176±21.67	186±30.49	205±33.19
730yrs. (n=5)	172.6±43.97	175.2±46.31	184.6±40.32

* Denotes significant 'P' valve ($P < .05$) when compared with fasting valve.

** Denotes significant 'P' valve ($P < .05$) when compared with preceeding and fasting valve.

EFFECT OF FAT CONSUMPTION

5 subjects 16% consumed high amounts of fat, 13 (42%) subject consumed in moderate amount, while the remaining 13 (42%) had a low daily fat consumtion. Basal STG levels were however not related to fat consumtion low fat intakers had the highest STG levels followed by high intakers. The difference between them was not statistically significant ($p > .05$) (Table 50).

On feeding single high cholesterol test lead all the groups showed rise in STG levels after one and three hours of feeding. But the rise was statistically significant in high and moderate fat consumers and insignificant in low fat consumer group.

Table 30

Table showing basal STG levels in high, moderate and low fat consumers of group A and changes in STG levels after single cholesterol test load in them.

Groups	Basal factors mg/dl	1 hr.after feedin mg/dl	3hrs.after feeding mg/dl
High (n=5)	148.8±32.3	160.6±40.04	179*±23.2
Moderate (n=13)	131.7±41.1	142.4±40.6	159.3±36.0
Low (n=13)	159.4±35.8	174.1±46.2	173.6±51.8

* Denotes significant P valve (P<.05) when compared with corresponding fasting valve.

EFFECT OF PHYSICAL ACTIVITY

23 subjects (74%) were doing moderate physical activity daily, 3 subjects (10%) did heavy physical work, while, remaining 5 subjects (16%) were sedentary in nature. Sedentary subjects had the highest basal STG level, as compared to moderate and heavy physical workers. However this difference was not statistically significant (Table 51).

On feeding single high cholesterol test load rise in STG levels was observed in all groups one and three hours after, but the magnitude of rise was more in moderate and heavy physical workes group, as compared to sedentary group. The rise was

highly significant (P<.01) in moderate physical workers group (Table 51).

Table 51

Table showing basal STG levels in heavy, moderate and sedentary subjects of group A and effect of feeding on STG levels in them.

Groups	Basal fasting	1hr.after feeding	3hrs.after feeding
Heavy (n=3)	154.3±39.57	194±84.04	190.66±69.97
Moderate (n=23)	139.2±36.46	151.52±35.34	162.06±36.98
Sedentary (n=5)	172.6±43.97	175.2±46.31	184.6±36.06

++ Denotes highly significant P valve (P<.01) when compared with fasting valve.

CHANGES OF STG IN GROUP B

This group had the second highest level of basal fasting serum triglyceride after diabitic subjects, however the difference was statistically insignificant (P>.05). On feeding single high cholesterol test lead, there was rise in STG level in 1 and 3 hours after feeding. 3 hours rise was statistically significant over fasting and 1 hour valve (Table 48).

EFFECT OF AGE

Lowest STG valve was observed in youngest subject, and this valve increased with age. Highest valve was observed in the subject of age groups of 41-50 years. In subjects 750 years, this value slightly decreased over preceeding group value. All STG valves were statistically significant ($P<.05$) over 21-30 year age group valve (Table 55).

On feeding single high cholesterol test load, all the age groups showed a rise in one and three hours valve, rise was statistically significant as shown in table 55.

Table 55

Effect of single high cholesterol fat test load on mean STG in various subgroup of group B

Age group	I Basal value mg/dl	II 1hr.after mg/dl	III 3hr.after mg/dl
21-30yrs. (n=12)	154.9±30.2	154.2±35.86	173.39±44.23
31-40yrs. (n=3)	225.3±65.30	227.6±75.14	243.3**±79.6
41-50yrs. (n=3)	268.3±21.8	281.6**±28.5	304**±36.8
750yrs. (n=2)	226±20	246.5**±13.5	276.5±6.5

* Denotes P valve is significant ($P<.05$) when compared with 21-30yrs. age group basal value by student to test.

** P valve is significant ($P<.05$) when compared with corresponding fasting value by paired t test.

*** Denotes P valve is significant (P/.05) when compared with fasting and preceedingx value.

EFFECT OF FAT CONSUMTION

5 subjects (25%) were high fat takers, while rest 15 subjects (75%) took low fat daily. High fat takers had a considerable high basal STG level, though this was not statistically significant (table 56).

On feeding single high cholesterol test load, both group showed rise in STG level 1 and 3 hours after feeding. Three hour rise was statistically significant over fasting valve in both the groups (table 56).

Table 56

Table showing basal STG values in high and low fat intakes subjects of group B, and effect of feeding single high cholesterol test load in them

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
High (n=5)	225±70.05	232.6±78-41	249.6±79.73
Low (n=15)	177.64±48.17	180.46±56.46	201.6±59.41

* Denotes significant P value (/.05) when compared corresponding fasting value.

EFFECT OF PHYSICAL ACTIVITY

16 subjects (80%) were doing moderate physical work daily while remaining 4 (20%) were sedentary in nature. Sedentary subjects had a considerable higher level of basal STG, but this was not statistically significant (Table 37) on feeding single high cholesterol test load sedentary subjects showed a significant rise of STG in first hour, But value was unchanged in moderate workers. After three hours both the groups showed statistically significant (Table 37).

Table 37

Table showing basal STG values in sedentary and moderate physical worker subjects of group B and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Sedentary (n=4)	217.0±51.15	235.75±63.44	230.75±37.86*
Moderate (n=16)	182.56±37.15	182.93±62.4	204.33±67.5*

* Denotes significant P value (P<.05) when compared with corresponding fasting value.

CHANGES IN SERUM TRIGLYCERIDE IN GROUP C

First degree relatives of patients of coronary artery disease had a higher basal STG level than healthy male volunteers (Table 48) though the difference was not statistically significant. On feeding single high cholesterol test load, initially there was a fall in STG level in first hour, a phenomenon not observed in any other groupl followed by a very minor rise in third hour value.

EFFECT OF FAT INTAKE

10 subjects (91%) consumed fat in moderate amounts daily, while the remaining one subject (9%) was a low fat taker. Moderate fat intakers had a slightly higher basal level of triglyceride as compared to low fat takers. (Table 99). On feeding single high cholesterol test load, 1 hour value showed a slight decrease in moderate fat intakers, while in low fat intaker, the value was unaffected. After three hours, in moderate fat consumers STG showed a further decline while in low fat taker, STG value rose tremendously.

Table 59

Table showing basal STC levels in moderate and low fat consumers of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=10)	185-1±56-13	182.9±50.01	179.6±62.70
Low (n=1)	171.0	171.0	256.0

EFFECT OF PHYSICAL ACTIVITY

9 subjects (81%) were doing moderate physical activity daily while the remaining 2 subjects (19%) did heavy physical work. Both groups had very near basal STC valves. On feeding single high cholesterol test load both the groups showed fall in STC levels in first hour. This fall continued in third hour in moderate group while in heavy workers, there was rise in STC level, three hour after feeding (Table 60). None of the change was statistically significant.

Table 60

Table showing basal STG valve in moderate and heavy physical worker subjects of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
High (n=2)	185±113.13	179±100.40	190.44±65.73
Moderate (n=9)	184±44.35	182.4±50.40	170.5±50.5

EFFECT OF SMOKING

3 subjects (27%) were smokers, while rest 8 subjects 33% were nonsmokers, smokers had a considerably higher basal level of STG as compared to nonsmokers. But the difference was statistically not significant.

On feeding single high cholesterol test load, botht the groups initially showed a fall in STG level in first hour, this fall continued in third hour in smokers, while in nonsmokers there was rise in STG level three hour after feeding. None of the changes were statistically significant (Table 61).

Table 61

Table showing basal STG levels in smokers and nonsmokers subject of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Smokers n=3	215.66±41.58	211.33±49.16	177.3±57.63
Non smokers n=3	171.87±54.63	170.7±56.05	190±53.31

CHANGES OF SERUM TRIGLYCERIDE IN DIABETIC SUBJECTS (GROUP D)

Diabetics had the highest basal serum triglyceride among all the groups (Table 48), among them type II or maturity onset diabetic had a higher basal STG then type I diabetics (Table 62).

On feeding single high cholesterol test load rise of STG levels was observed one and three hours after feeding. Three hours rise was statistically significant over basal (Table 48).

Table 62

Table showing basal STG levels in type I and type II diabetics and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3 hrs.after feeding mg/dl
Type I (n=5)	151.3±6.59	146.6±18.8	180.0±43.2
Type II (n=5)	256.2±52.8**	275±36.9*	270.6±41.74*

* Denotes significant 'P' valve when compared with fasting valve.

** Is Significant 'P' valve (P/.05) when compared with basal valve of type I diabetics by student 't' test.

Feeding in type I diabetics resulted in fall in STG level in first hour followed by rise in third hour, while in type II diabetics, maximum rise in STG levels occured in first hour and three hour valve showed a slight fall. Rise of 1 and 3 hour was statistically significant (Table 62).

EFFECT OF FAT CONSUMPTION

6 subjects (75%) consumed low fat diet daily while 2 subjects (25%) consumed high fat diet daily. High fat consumers had a very high basal STG level as compared to low fat takers.

On feeding both the group showed rise in STG levels one hour after feeding, while this rise continued in third hour in low fat takers, moderate consumers showed a fall in STG levels.

Table 63

Table showing basal STG valve in low and moderate fat consumers of group D and effect of single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Low (n=6)	186-0±43.04	199.1±70.47	216.5±48.93
Moderate (n=2)	259-5±101.11	310.0±162.63	297.0±53

All the changes were statistically insignificant in table 63-

CHANGES IN VERY LOW DENSITY LIPIDS (VLDL)

Highest level of VLDL was observed in diabetics followed by healthy females and first degree relatives of patients of coronary heart disease. Healthy males had the lowest value among all (Table 65), on feeding single high cholesterol load, increase in VLDL levels were observed in all groups three hours after feeding.

CHANGES IN GROUP A (HEALTHY MALES)

Basal VLDL levels were 29.19±7.7 mg/dl, on feeding it increased significantly 1 and 3 hours after. Rise was statistically significant (P<0.005) (Table 65).

Table 65

Effect of single high cholesterol test load on mean VLDL levels in group A, B, C and D.

Group	Basal fasting mg/dl I	1hr. after test load mg/dl II	3hrs. after test load mg/dl III
A (n=31)	29.19±7.78	31.53±8.96	33.71.71±8.66
B (n=20)	38.27±11.18	38.66±12.93	42.73±14.0
C (n=11)	36.7±10.1	36.3±10.5	37.3±12.73
D (n=8)	40.87±12.55	45.37±19.95	47.32±12.18

* Denotes significant 'P' value when compared with fasting value.

** Denotes highly significant 'P' value ($P < .005$) when compared with fasting and fasting and preceeding valve.

EFFECT OF AGE

Basal VLDL levels increased progressively with rising age till 30 years, after which in subjects of 730 years of age, a slight fall in levels was observed (Table 66) on feeding single high cholesterol test load rise in VLDL levels was observed in all age groups 1 and three after, but the rise was maximum in youngest subjects, while the effect of feeding was blunted with the rise of age. Thus only in 20-30 years old subjects the rise was statistically significant (Table 66).

Table 66

Effect of single high cholesterol test load on mean VLDL levels in various age subgroups of Group A

Group	I Basal valve mg/dl	II 1hr.after mg/dl	III 2hrs.after mg/dl
21-30yrs. (n=14)	24.9±6.42	26.97±6.69 *	30.0±5.57 **
31-40yrs. (n=7)	29.3±6.04	34.11±10.22	33.48±11.19
41-50yrs. (n=5)	35.6±3.8	37.2±5.45	41.0±5.93
730yrs.	34.2±7.86	35.04±8.28	36.92±7.21

* Significant 'P' valve ($P < .05$) when compared with fasting valve.

** Significant 'P' valve ($P < .005$) when compared with fasting.

EFFECT OF FAT CONSUMTION

5 subjects (16%) were high fat consumers while 13 subjects (42%) each were moderate and low fat consumers respectively fat intake did not seemed to have any significant effect on basal VLDL levels. Thus low fat intakers had a higher basal VLDL level as compared to high and moderate fat intakers. On feeding single high cholesterol test load all of them showed rise of VLDL level, but statistically rise was only significant in high and moderate fat consumers, three hours after feeding (Table 67).

Table 67

Table showing basal VLDL level in high, moderate and low fat consumers of group A and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr. after feeding mg/dl	3hrs. after feeding mg/dl
High n=5	29.76±6.46	30.92±9.03	35.92±4.49 *
Moderate n=13	26.29±8.31	28.49±8.13	31.85±7.19 **
Low n=13	31.87±7.17	34.81±9.25	34.72±10.35

** Denotes highly significant 'P' valve ($P < .005$) when compared with fasting valve.

EFFECT OF SMOKING

25 subjects (80%) were smokers while remaining 6 were nonsmokers. Nonsmokers had a slightly higher level of basal VLDL which was statistically insignificant. But on feeding smokers showed a greater magnitude of rise in VLDL levels as compared to nonsmokers, though it was not statistically significant (Table 68).

Table 68

Table showing basal VLDL level in smokers and nonsmokers and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Nonsmokers (n=25)	29.45±8.08	31.72±9.38	32.43±7.78
Smokers (n=6)	28.1±6.93	30.75±7.05	34.06±5.59

EFFECT OF PHYSICAL ACTIVITY

23 subjects (74%) were moderate workers 3(10%) were heavy workers while the remaining 5(16%) were sedentary. Sedentary subjects had the highest basal VLDL levels as compared to heavy and moderate

workers. All the subjects showed a rise in VLDL levels 1 and 3 hours after feeding single high cholesterol test load though the rise was not significant in any (Table 69)

Table 69

Table showing basal VLDL levels in heavy, moderate and sedentary workers of subjects of group A and effect of single high cholesterol test load in them

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Sedentary (n=5)	34.52±8.79	35-04±9.26	36-92+7.21
Moderate (n=23)	27-81±7.35	29.79±7.41	32.43±7.38
Heavy (n=3)	30.86±7.91	38.73±16-90	38.13±13.9

CHANGES OF CLDL IN GROUP B (HEALTHY FEMALES)

Basal VLDL level was 38.27±11.18 mg/dl on feeding single high cholesterol test load it showed a rise in 1 and three hours after three hours rise was statistically significant (Table 65).

EFFECT OF AGE

With increasing age a rise in basal VLDL level was observed. Basal levels were maximum in the age group of 40-50 years and then after a slight fall was

observed. On feeding statistically significant rise of VLDL was observed in all age groups (Table 71).

Table 71

Effect of single high cholesterol test load on mean VLDL level in various age related subgroups of Group B

Group	I Basal valve mg/dl	II 1hr.after test load mg/dl	III 3hrs.after test load mg/dl
21-30yrs. (n=12)	31.6±6-18	30.8±6-87	34.6±8.49*
31-40yrs. (n=3)	45±10.6	45.5±12.2	48.66±12.9**
41-50yrs. (n=3)	53-66±4.36	56-33±5.71**	68.8±7-36***
7/50 yrs. (n=2)	49.2±4.0	49.3±2.7	55.3±1.3

* Denotes significant P valve (P<.05) when compared with preceeding valve.

** Denotes significant P valve (P<-05) when compared with fasting valve.

*** Denotes significant P valve (P<.05) when compared with preceeding and fasting valve.

EFFECT OF FAT CONSUMPTION

15 subjects (75%) were low fat consumers. High basal VLDL levels were observed in high fat consumer groups. On feeding single high cholesterol load, no effect on VLDL level was observed in low

fat consumers, while high fat consumers showed a higher rise in first hour after feeding and this rise was sustained in third hour (Table 72).

Table 72

Table showing basal VLDL levels, in high and low fat consumer subject of group B and effect of single high cholesterol test load in them.

Group	Basal fasting	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Low (n=15)	36.07±9.56	36.1±11.28	36.14±10.8
High (n=5)	45±4.01	46.52±15.68	46.52±14.02

EFFECT OF PHYSICAL ACTIVITY

16 subject (80%) were moderate physical workers while remaining (20%) were sedentary. Sedentary subjects had a considerable higher level of basal VLDL as compared to moderate workers. On feeding single high cholesterol test load both the groups showed a rise in VLDL level 1 and 3 hours after, but the rise was much more marked and statistically significant in sedentary subjects (Table 73).

Table 73

Table showing basal VLDL levels in moderate and sedentary workers of group B and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=16)	39.96±11.18	36.59±12.47	40.87±13.50
Sedentary (n=4)	43.4±10.23	47.15±12.68	50.15±11.57

CHANGES OF VLDL IN GROUP C

First degree relatives of patients of coronary aretery disease had a basal mean VLDL level of 36.7±10.1mg/dl. On feeding single high cholesterol test load, VLDL levels showed a slight fall in first hour followed by a slight rise in third hour. None of the change was statistical (Table 65) significte.

EFFECT OF FAT CONSUMTION

10 subjects (91%) were moderate fat consumers, while 1 subject (9%) was low fat consumer, moderate fat consumers had a higher basal level of mean VLDL as compared to low fat consumers. On feeding single high cholesterol test load in them, moderate fat

consumers showed a fall in mean VLDL level in 1 and 3 hours, while in low fat consumer VLDL levels rose in three hours. None of the change was statistically significant (Table 75).

Table 75

Table showing basal mean VLDL levels in moderate and low fat consumers subjects of group C and effect of feeding single high dose cholesterol in them.

Group	Basal mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=10)	37-02±11.22	36-50±11.60	35.42±13.27
Low (n=1)	34.2	34.2	51.2

EFFECT OF PHYSICAL WORK

9 subjects 82% were moderate workers while remaining 2 subjects (18%) were heavy workers. Moderate workers had a higher mean VLDL level as compared to heavy workers and on feeding single high cholesterol test load heavy workers showed a fall in VLDL level in 1 and 3 hours, while moderate workers showed a rise in VLDL levels 3 hours after feeding (Table 77).

Table 77

Table showing basal mean VLDL level in moderate and heavy worker subject of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Moderate (n=9)	36-8±8.87	36-48±10.08	38.04±13.91
Heavy (n=2)	36-6±22.62	35.8±20.08	34.1±14.28

CHANGES OF VLDL IN GROUP D (DIABETIES)

Diabetic subjects had the highest level of mean basal VLDL among all the groups. Type II diabetais exhibited a higher basal level than type I diabetics. On feeding single high cholesterol test load, a rise in VLDL level was observed 1 and 3 hours after feeding. Three hours rise was statistically significant (Table 65). Feeding effect was much more pronounced in type II diabetics than type I where actually a fall in VLDL level 1 hr. after feeding was observed. Rise in type II diabetics was statistically significant 1 and 3 hours after feeding (Table 78).

Table 78

Table showing basal mean VLDL levels in type I and type II diabetics and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr. after feeding mg/dl	3hrs. after feeding mg/dl
Type I (n=3)	30.2±1.3	29.3±3.77	36±8.64
Type II (n=5)	47.2±10.5	53.0±17-3	34-12±8.34

* Denotes significant 'P' value when compared to fasting value.

EFFECT OF FAT CONSUMPTION

6 subjects (85%) were low fat consumers, while the remaining 2 subjects (20%) were moderate fat takers. Basal mean VLDL levels was much higher in moderate fat takers than low fat taker. On feeding single high cholesterol test load, both the groups shows a rise in VLDL levels in first hour. While this rise continued into three hours in low fat takers, moderate fat consumers showed a slight fall in VLDL levels after three hours.

Table 72

Table showing mean basal VLDL levels in low and moderate fat consumers diabetic subjects and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Low (n=6)	37-2±8.60	39.83±14.09	43.3±10.72
Moderate (n=2)	51-9±20.22	62±32.52	59.4±14.99

None of the changes was statistically significant.

CHANGE IN LDL CHOLESTEROL

Diabetics had the highest level of basal mean LDL cholesterol, it was followed by females and first degree relatives of patients of coronary artery disease. On feeding single high cholesterol load, a fall in LDL level was observed ingroup A, B and D, while in group C level remained unchanged. After three hours of feeding LDL still showed a declining trend in group A, and B while in group C levels started rising, but it did not cross fasting valve. None of the change was statistically significant (Table 81).

Table 31

Effect of single high cholesterol test load on mean LDL levels in group A, B, C and D

Group	I Basal mg/dl	II 1hr.after mg/dl	III 3hrs.after mg/dl
A (n=31)	88.90±37.0	79.0±30.93	78.97±31.23
B (n=20)	99.7±29.71	92.7±31.11	90.89±33.04
C (n=11)	98.39±24.35	98.3±37.7	97.9±32.94
D (n=8)	119.27±30.79	105.75±24.18	115.01±23.8

CHANGES OF LDL CHOLESTEROL IN GROUP A

Basal levels of LDL-c were 88.90±30.0 mg/dl. After feeding it decreased in 1 and 3 hours. Out of total of 31 subjects, 16(52%) showed a fall in LDL-c level one hour after feeding, six subjects (19%) did not revealed any effect of feeding, while the remaining 9 subjects (29%) showed a rise in LDL level within one hour. These responses were respectively termed as LA III, LA II and LA I.

On analysing the feeding effect after 3 hours it was observed that in subjects which showed a fall in first hour (LA III), only in (25%) did this falling trend persisted, while in the rest 12 (75%) a rise in LDL valve was observed over 1 hour valve, and in some cases 3 hours valve exceeded fasting valve. All the changes of 1 and 3 hours were statistically significant (Table 32). These subjects also had a higer basal mean LDL levels than others.

Table 32

Table showing three different type of behavior observed in LDL-c level after feeding single high cholesterol test load in subjects of group A.

Group	Basal mg/dl	1hr.after mg/dl	3hrs.after mg/dl
LA-I (n=9)	80.13±27.64	99.52±29.83*	87.9±22.66
LA-II (n=6)	71.15±35.36	72.11±34.99	97.41±29.36
LA-III (n=16)	100.48±39.98	69.98±26.13*	82.02±33.84**

* Denotes significant 'P' valve ($P<.05$) when compared with preceeding valve.

** Denotes significant 'P' valve ($P<.05$) when compared with fasting and preceeding valve.

In subject in whom a rise in LDL-c level was observed 1 hr. after feeding (LA-I), only in 2 subject (22%) did this riseing trend persisted after three hours of feeding, rest 7 subjects (78%) showed a fall in LDL-c levels over 1 hr. levels, however changes of 1 hr. and three hours were statistically significant (Table 82).

EFFECT OF AGE

A decreasing level of mean basal LDL-c level was observed with increasing age, thus the youngest age group subjects (21-30 yrs) had the highest mean basal LDL level of 108.5±38.39 mg%. While the eldest subjects of 71-80 years of age had the lowest basal LDL levels (Table 83). On feeding single high cholesterol test load, a fall in LDL-c level was observed in all age group, but the magnitude of the fall decreased with rising age, none of the change was statistically significant (Table 83).

Table 83

Effect of single high cholesterol test load on mean LDL levels in various age related subgroups of group A.

Group	I Basal valve mg/dl	II 1hr.after mg/dl	III 3hrs.after mg/dl
21-30yrs. (n=14)	108.5±38.89	93.81±31.11	93.47±33.49
31-40yrs. (n=5)	74.59±19.89	69.97±30.24	72.35±25.09
45-50yrs. (n=5)	73.04±24.86	62.32±12.56	61.4±20.64
50yrs. (n=5)	69.98±28.92	66.86±20.92	65.18±16.0

EFFECT OF FAT CONSUMTION

5 subjects (16%) were consuming high daily fat, while out of remaining 26, 13 each were consuming fat in moderate and low amounts respectively.

High fat consumers were having considerably high level of basal mean LDL, followed by moderate and low fat consumers. On feeding single high cholesterol test load all the groups showed a fall in LDL levels after 1 hour. In high and moderate

fat consumers falling trend continued in third hour, while in low fat consumers there was rise in LDL level equalling fasting level. None of the change was statically significant (Table 84).

Table 84

Table showing basal mean LDL-c level in high moderate and low fat consumers subjects of group A and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
High (n=5)	139.34±48.26	107-58±26.21	102.98±22.43
Moderate (n=13)	87.96±21.99	83.56±29.96	75.54±24.42
Low (n=13)	70.39±26.97	63.32±25.06	70.7±23.7

EFFECT OF SMOKING

6 subjects (19%) were smokers, while remaining 25%(81-1) were nonsmokers. Smokers had a considerable higher level of basal LDL-c than nonsmokers. On feeding single high cholesterol test load both the groups showed a fall in LDL level 1 hour after feeding, while this falling trend persisted in nonsmokers as revealed by three hours value, smokers showed a rise in LDL levels after three hours, none of the change was statistically significant (Table 85).

Table 85

Table showing basal LDL-c levels in smokers and nonsmoker subjects of group A and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Smokers (n=6)	120.8±40.48	146.88±24.47	112.5±32.83
Non smokers (n=2)	81.1±32.58	72.04±28.71	70.7±25.19

EFFECT OF PHYSICAL ACTIVITY

23 subjects (74%) were moderate workers, 3 subjects (10%) were heavy workers, while remaining 5 subjects (16%) were sedentary in habits. Sedentary workers had the lowest basal mean LDL-c level. On feeding single high cholesterol test load, all the groups showed fall in LDL level after 1 hour, but the fall was minimum in sedentary subjects and maximum in heavy workers. After three hours heavy workers showed a increase in LDL levels over 1 hour value. In the rest of the group fall of LDL continued into third hour. None of the change was statistically significant (Table 86).

Table 86

Table showing basal LDL-c level in sedentary moderate and heavy workers subjects of group A and effect of feeding single high dose cholesterol in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Sedentary n=5	69.98±32.33	66.86±23.39	65±17.84
Moderate n=23	94.48±38.25	84.43±31.54	82.4±34.24
Heavy n=3	75.46±27.37	55.8±26.79	79.33±17.95

CHANGES OF LDL CHOLESTEROL IN GROUP B (HEALTHY FEMALES)

Group B had a basal mean LDL level of 99.7±29.71 mg/dl. On feeding single high cholesterol test load, the valve of LDL showed a fall in level after one and three hours none of the change was statistically significant (Table 91). Three different types of responses were observed after feeding. In 11 subjects (55%) a fall in LDL level was observed 1 hr. after feeding, this type of response was turned on LD-III. In 5 cases (25%) a rise in LDL level was observed after 1 hr. of feeding. This type of response was

termed as LB-I and in the remaining 4 cases (20%) no change in LDL valve was observed after feeding this was termed as LB-II type of response (Table 88).

Table 88

Table showing three different type of feeding induced changes in LDL-c observed in subjects of group B.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.feeding mg/dl
LBI (n=5)	91.52±19.3	103.72±19.69*	98.96±35.64
LB-II (n=4)	111.75±22.85	112.02±22.62	109.99±21.98
LB-III (n=11)	99.11±35.67	80.90±34.04*	80.31±33.49**

* Denotes significant P valve (P<.05) when compared with fasting valve.

** Denotes significant P valve (P<.05) when compared with fasting and preceeding valve.

In group LB-I after 3 hour, rise of LDL persisted only in two subjects out of five (40%) rest 3 subjects (60%) showed a fall in LDL level as compared to 1 hour levels. In group LB-III 6 subjects out of 11 (55%) exhibited a continuation of the first hour pattern, i.e. fall in LDL levels, while the remaining 5 subjects (45%) showed a rising

trend. In this group changes of 1 and 3 hours were statistically significant (Table 88).

EFFECT OF AGE

LDL-c levels increased with increasing age up to 50 years, then after in subjects of 750 years of age a fall in LDL levels was observed. On feeding single high cholesterol test load, a fall in LDL level was only observed in youngest subjects i.e. 21-30 years age group, after 1 hour, while remaining group showed a minor rise in LDL-c level in the same duration. After three hours, fall of LDL-c persisted in 21-30 years. Age group, while in the rest, a rise,or no change was evident. Three hours changes of age group 21-30 years and 750 years were statistically significant (Table 89).

Table 89

Effect of single high cholesterol load on mean LDL level in various age related subgroup of group B.

Group	I Basal valve mg/dl	II 1 hr.after mg/dl	III 3hrs.after mg/dl
21-30yrs. (n=12)	91.24±28.2	77.1±26.2	71.7±22.2
31-40yrs. (n=5)	111.8±21.4	112.6±21.3	122.6±25.92
41-50yrs. (n=3)	129.3±19.4	130.4±6.55	127.2±17-86
51-60yrs. (n=2)	88.4±16.4	99.7±8.3	103.2±8.8

* Denotes significant 'P' valve when compared with fasting valve.

** Denotes significant 'P' valve when compared with fasting and preceeding valve.

EFFECT OF FAT CONSUMTION

15 subjects (75%) were low fat consumers while remaining 5 (25%) were high fat takers. High fat taker had a high basal LDL level than low fat consumers. On feeding single high cholesterol test load, low fat user showed a fall in LDL-c level in 1 and 3 hours, while a very little change was observed in high fat takers (Table 90).

Table 90

Table showing basal mean LDL-c level in high and low fat consumers subjects of group B and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
High n=5	122.4±25.40	118.8±21.08	120.82±27.32
Low n=15	91.88±27.59	84.34±28.42	81.78±28.26

EFFECT OF PHYSICAL ACTIVITY

Sedentary subjects had a higher basal mean LDL-c level than moderate workers. On feeding single high cholesterol test load, sedentary subjects showed a rise in LDL level 1 and 3 hours after feeding, while moderate workers showed a fall in the same duration (Table 91).

Table 91

Table showing basal LDL-c levels in sedentary and moderate workers of group B and effect of feeding high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Sedentary (n=4)	107.5±26.28	113.6±17.42	120.85±21.68
Moderate (n=16)	97.65±30.84	87.66±31.48	83.38±29.7

CHANGES OF LDL CHOLESTEROL IN GROUP C

Basal mean LDL- levels in first degree relative of patients of coronary artery disease was 98.39±24.35 mg/dl. Practically no effect in LDL levels was observed 1 and 3 hours after feeding high cholesterol test load.

6 out of 11 subjects (55%) demonstrated rise in LDL-c level 1 hour after feeding, while the rest showed a fall in LDL-c level in the same duration. These responses were termed as LCI and LCIII respectively (Table 93). After three

hours in only three subjects exhibiting LCI type of responses did the rise continued, while in three there was fall in LDL-c level. Similarly in LCIII type of responders four subject (80%) showed a falling trend in third hour, while in remaining the a rise in LDL level was observed.

Table 93

Table showing basal mean LDL level in LCI and LCIII type of responders and effect of feeding in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
LCI (n=6)	102.13±32.6	124.7±31.85***	112.4±37.03
LCIII (n=5)	93.9±15.84	66.6±18.71*	80.4±23.89

*** Highly significant P (P<.001) when compared with fasting valve.

* Significant P (P<.05) when compared with fasting value.

EFFECT OF FAT CONSUMTION

10 subjects (91%) were moderate fat consumers, while remaining 1 (9%) was low fat taker. Basal LDL-c levels were practically same in both the groups. After feeding moderate fat consumers, showed a slight fall in

LDL-c level in first hour followed by rise in third hour. While in low fat consumer, a rise in LDL-c level in first hour was observed.

Table 94

Table showing basal LDL level in moderate and fat consumer subjects of group C and effect of feeding single high cholesterol test load in them.

Group	Basal fasting mg/dl	1hr. after feeding mg/dl	3hrs. after feeding mg/dl
Moderate (n=10)	98.35±26.92	95.48±40.61	99.83±35.80
Low (n=1)	98.8	125.8	78.8

after feeding was observed, it was followed by a fall in third hour (Table 94).

CHANGES IN LDL-C IN GROUP D

Group D showed the highest amount of basal mean LDL level among all of the group. Type II diabetics showed a significantly higher level of basal LDL than type I. On feeding single high cholesterol test load, type I diabetics, showed a rising trend of LDL level after 1 and three hour.

while in type II a fall in levels was observed 1 and 3 hours after, All the changes were significant in both the groups.

Table 96

Group	Basal fasting	1hr.after feeding	3hrs.after feeding
Type I (n=3)	63±3.9.0	77.5±7.88	95±11.4
Type II (n=5)	152.8±23.6	122.7±3.96	127.02±21.2

* Denotes significant 'P' valve when compared with fasting levels.

** Denotes significant P valve (P<.05) when compared to fasting and preceeding value.

EFFECT OF FAT CONSUMPTION

Moderate fat consumers showed a higher basal LDL level than low fat consumers. On feeding both group showed a fall in LDL level in first hour which was much more marked in moderate fat intakers. After three hours, low fat intakers exhibited a rising LDL level over 1 hour, while in moderate fat consumers level were unaffected (Table 97).

Table 97

Table showing basal LDL level in low and moderate fat consumers and effect of feeding in subjects of group D.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Low (n=6)	100.33±43.13	99.58±25.22	112.01±28.93
Moderate (n=2)	175.9±9.9	124±0	124±9.0

CHANGES IN LDL/HDL RATIO

Basal mean LDL/HDL ratio was within normal limits in all the groups (i.e.<3). Feeding single high cholesterol test load reduced the ratios in group A and B after 1 and 3 hours. But in group C (first degree relative of CAD) ratio increased in the first hour and again decreased in third hour. While in diabetics (Group D) ratio decreased in first hour and increased in third hour (Table 99).

Table 99

Table showing basal LDL/HDL ratio in groups A, B, C, and D and effect of feeding on it.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Group A (n=31)	1.97±0.83	1.77±0.73	1.77±0.74
Group B (n=20)	2.48±1.49	2.10±0.97	2.00±0.97
Group C (n=11)	2.43±0.96	2.47±1.15	2.21±0.90
Group D (n=8)	2.5±0.86	2.26±0.41	2.74±0.61

CHANGES IN LDL/DDL RATIO IN GROUP A

3 subject (9.6%) had basal LDL/HDL ratio in excess of 3, 12 subjects (38%) showed a rise in LDL/HDL ratio 1 hour after feeding. While 11 subjects (35%) showed increase in LDL/HDL ratio after 3 hours over basal. In rest the ratio decreased after feeding. Highest LDL/HDL ratio was observed in youngest subjects. This ratio progressively decreased with age. Feeding induced fall in LDL/HDL ratio in all age groups in first hour, but the magnitude of the fall decreased with age (Table 100).

Table 100

Showing distribution of LDL/HDL ratio in different age groups of group A.

Groups	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
21-30yrs. n=14	2.38±0.87	2.16±0.82	2.07±0.79
31-40yrs. n=7	1.73±0.62	1.56±0.53	1.64±0.44
41-50yrs. n=5	1.58±0.75	1.25±0.35	1.44±0.77
7 50yrs. n=5	1.56±0.83	1.47±0.64	1.44±0.52

CHANGES IN LDL/HDL RATIO IN GROUP B

5 subjects (25%) had an abnormal basal LDL/HDL ratio (i.e. 7 5). Six subjects (30%) showed a rise in ratio 1 hour after feeding, while in the rest feeding induced fall in the ratio. Highest basal ratio was observed in the age group of 41-50 years. On feeding single high cholesterol test load, younger subjects showed a fall in ratio after 1 hour, while older subjects (7 40 years) showed a rising trend after 1 hour (Table 101).

Table 101

Showing distribution of LDL/HDL ratio in different age groups of group B and effect of feeding in them.

Groups	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
21-30yrs. n=12	2.50±1.92	1.68±0.88	1.45±0.57
31-40yrs. n=3	2.01±0.10	1.97±0.12	1.92±0.40
41-50yrs. n=3	3.15±0.75	3.51±0.83	3.43±0.69
7 50yrs. n=2	2.12±0.37	2.73±0.03	3.28±0.34

* Denotes significant P value when compared with fasting value.

CHANGES IN LDL/HDL RATIO IN GROUP C

In only 2 subject (18%) basal LDL/HDL ratio was abnormal 5 subjects (45%) showed a increased ratio after 1 hour feeding. On the whole group C showed slight increased in ratio 1 hour after feeding, which fall back to below fasting ratio after 3 hours.

CHANGES OF LDL/HDL RATIO IN GROUP D (DIABETICS)

Diabetics had the highest basal LDL/HDL ratio. Among diabetics, type II diabetic subjects showed basal ratio of 7. On feeding, there was progressive increase in ratio in type I diabetics after 1 hour

and 3 hours, while in type II diabetics, there was initeed fall in first hour, followed by rise in third hour.

Table 102

Table showing basal LDL/HDL ratio in type I and type II diabetics, and effect of feeding in them.

Group	Basal fasting mg/dl	1hr.after feeding mg/dl	3hrs.after feeding mg/dl
Type I n=5	1.47±0.36	1.86±03	2.46±3.36***
Type II n=5	3.14±0.27	2.5±0.34	2.9±0.65

*** Highly significant P value (P<.001) when compared with fasting and preceeding value.

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DISCUSSION

DISCUSSION

Estimation of diet induced changes in lipid lipoprotein profile and its relevance in predicting an individual risk for future atherosclerosis still remains an unexplored field.

Drastic individual variability in assimilating a cholesterol fat test load is the main reason why till now no reasonable cholesterol tolerance test could be devised. Individual also respond differently to single feeding and prolong feeding, thus this still deepens the mystry, whether a single dose or a prolong feeding programme could be a better screening procedure.

The possibility that the particular response of an individual to single high cholesterol test load may be the determinant of the development of the atherogenic process in him, has formed the basis of the present work.

This work has been conducted on healthy male and female volunteers of age 20-60 years, healthy first degree relatives of patients of

coronary artery disease and diabetic patients.

SERUM TOTAL CHOLESTEROL

The basal serum total cholesterol was 164.4±36.98, 185.2±36.2, 178.72±30.17 and 206.87± 64.75 mg/dl of group A, B, C and D respectively.

Group A, B and C had basal STC within normal limits as set by lipid research clinics, however diabetics (group D) subjects had a 10% more basal STC level than normal subjects of the same age group. In healthy males (group A) highest basal levels was found in young subjects of 20-30 years, this may be because of high fat consumed and belonging to better socioeconomic status than older age person. However in healthy females (Group B) basal STC increased with age till 30 years and then after it showed a slight fall, this may be because of hormonal changes and repeated pregnancies associated with increasing age.

On feeding single high cholesterol test load consisting of 800 mg of egg cholesterol, a fall in STC after 4 hour was observed in majority

of subjects of group A, B, and type II diabetics, while in type I diabetics there was rise in STC level and in group C levels were more or less unaffected. After three hours, the levels of STC have started increasing, but still they were below basal levels in group A and B, while in group C and type I diabetics they were well above the fasting levels.

Thus on the basis of post prandial response the healthy population can be divided in three groups. The majority of the population shows a fall in serium total cholesterol and LDL levels, 1 hour after feeding, in a minority STC level start rising 1 hour after feeding, while in the remaining these in no change. In contrast to these results, majority of the subjects who were predisposed to the risk of CHD showed a rising trend of STC. Similarly all type I diabetics showed a rising trend of STC. In the past Nikkila et al. 1962 and Richard S, Haval 1957 have also reported fall in STC level after feeding. The explanation for this fall could be related to the suppression of LDL receptors after over night fasting (Medical clinics of North America Vol. 66 No.2

March 1982 page 344). When fat cholesterol load is given LDL receptors are stimulated by as yet some undefined hormonal or neurogenic reflexes, in anticipation of the cholesterol load that will enter the circulation. Large amount of LDL from intra-vascular compartment shifts intracellularly, resulting in an acute fall in serum LDL and STC levels after 1 hour. The cholesterol levels slowly increase after 3 hours as a result of the absorbed cholesterol and the reverse intravascular movement of LDL that had entered the tissues earlier.

In diabetics, majority of healthy relatives of patients of CAD and minority of healthy populations the increase of STC and LDL could be explained by some inherent biochemical block in anticipating and assimilating a cholesterol load.

HDL CHOLESTEROL

The basal HDL level were 46.07±6.34, 47.2±15.0, 43.5±11.3, and 47±8.27 mg/dl of group A, B, C and D respectively. These level were all within normal limits set by lipids research clinics. However group C subjects, i.e. first degree relation of patients of coronary artery disease, had comparitively lower level of HDL than their healthy counterpart subjects of group A. Age seems to have no effect on basal HDL level. Female sex had comparitively higher level of fasting HDL. On feeding single high cholesterol test load, no appreciable changes in HDL were observed after one hour. After three

hours, diabetic subjects (Group D) showed a significant fall in HDL levels, in other no significant changes were observed. Feeding induced fall in HDL level in diabetics was associated with rise of STC and LDL.

SERUM TRIGLYCERIDE

The basal STG levels were 146.0±38.7, 109.54±56.35, 183.8±50.93 and 204.37±62.7 mg/dl respectively of group A, B, C and D. All the groups had much higher STG valve than set by lipid research clinics. Diabetics had the highest level of basal STG.

Healthy females had a much higher value of STG than healthy males. Basal STG value tended to increase with rising age.

After feeding single high cholesterol test load, STG levels tended to increase 1 and 3 hours post prandially in all the groups, except first degree relatives of patients of coronary artery disease where in first hour there was actually a slight fall in mean STG level, followed by very insignificant rise in third hour. Maximum rise of STG levels was observed in diabetics.

The peak level of STG in our study was observed at the end of third hour.

Barritt (1956) Brown et al. (1961) and Angervall (1964) reported peak STG levels, four hours after feeding in healthy subjects.

Barritt 1956 reported peak of STG after seven hours in subjects of IHD, in our study first degree relating of patients of CAD had a delayed peak infact the STG started rising after 3 hours. end of 3 hours. Sklarin et al. (1961) and Mehra et al. (1983) also reported delayed and sustained rise of STG levels in diabetic subjects

VLDL CHOLESTEROL

Changes in VLDL were essetnaly similar to those observed in STG.

LDL CHOLESTEROL

Basal LDL valves were 88.90±37.0, 99.7±29.71, 98.39±24.25 and 119.27±30.79 mg/dl of group A, B, C and D respectively. These values were within limits set by lipid research clinics. Healthy females and diabetic subjects had a higher level of basal LDL

valves as compared to healthy males. First degree relatives of patients of CAD had a lower level of basal mean LDL levels than their healthy counterparts. Age seemed to have no appreciable effect on basal LDL levels.

Feeding single high cholesterol load resulted in decrease in LDL levels in healthy male, female and type II diabetic subjects, while no appreciable change was observed in first degree relatives of patients of coronary artery disease, in type I diabetics the levels actually started rising after feeding. After three hours the LDL levels were still declining in group A and B, levels were increasing in group D while they were unaffected in group C. The explaination for the observed phenomenon is the same as that for STC.

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CONCLUSIONS AND SUMMARY

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In the present work 31 male healthy volunteers, 20 female healthy volunteers, 11 male and female young healthy first degree relatives of patients of CAD and 8 diabetic subjects were studied to see the response of an individual to stress of single high cholesterol test load, in healthy, subjects at risk and in diseased patients. Following conclusions were drawn from the present study.

1. Three types of responses in behaviour of STC was observed 1 hour after feeding single high cholesterol test load. In the majority of healthy males and females and type II diabetics a fall in STC was observed, in minorty of these subjects plus majority of first degree relatives of CAD and type I diabetics showed a rise in STC level 1 hour after feeding, while in the remaining no change was observed.

2. The magnitude of fall in STC level declined with increasing age in all groups.

3. After 3 hours, STC values though showed a rise over 1 hour value but was still well

below fasting levels in Group A, Group B and Group D, while in Group C they crossed the fasting value.

4. Smokers had a significantly higher level of basal serum total cholesterol as compared to nonsmokers.

5. High daily fat consumers had a higher basal serum total cholesterol as compared to low fat consumers.

6. Basal HDL levels were much higher in young age females than males of the same age group.

7. First degree relatives of patients of CAD had a much lower level of HDL as compared to subjects of same age group without any risk factor.

8. Single high cholesterol feeding induced little change in HDL level 1 and 3 hour after feeding.

9. Highest basal levels of serum triglyceride was observed in diabetic subjects. First degree relation of patients of CAD also had a much higher level of basal STG as compared to healthy males of the same age group.

10. A progressive rise in STG level was observed 1 and 3 hour after feeding single high cholesterol test load in all the group.

11. The feeding induced rise in STG was maximum in diabetic subjects.

12. First degree relatives of patients of CAD showed a delayed rise in STG valve after feeding.

13. Changes in VLDL and LDL were exactly similar to that observed in STG and STC respectively.

14. A favourable shift in LDL/HDL ratio after feeding was observed in Group A, B and D while in Group C this ratio changed unfavourably on feeding.

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APPENDIX

Group A

Healthy - Males

S.No.	Name	Age (years)	Weight (kg.)	Height (cm.)
1	Dr.I.B.Singh	29	70	172
2	" Dilip Kumar	29	67	160
3	" Shaklani	24	62	162
4	" Mukul Gupta	26	60	158
5	" C.C.Sethi	27	74	172
6	" Sunil Ghai	28	70	174
7	" Sushil Nagsia	58	58	162
8	" Anil Garg	29	72	160
9	" Rajesh Tiwari	27	58	158
10	" D.K.Singh	26	62	164
11	" Subhas Ceel	26	70	164
12	" M.Chaturvedi	25	68	172
13	Mr.Ravindra Bhatt	30	68	168
14	Dr.I.K.Sharma	27	62	168
15	Mr.O.P.Shukla	38	60	162
16	" P.S.Sachan	38	60	164
17	" Ram Singh	35	52	160
18	" Mr.Gajraj	35	54	164
19	" Babu Lal	35	52	165
20	" Bhart	31	56	158
21	" Dhani Ram	38	70	174
22	" Mr.Manzoor	50	55	155
23	" Bare Lal	50	50	156
24	" Kali Charan	50	54	155
25	" J.F.Maher	46	85	160
26	" I.Ahmed	50	50	159
27	" Saru	65	50	162
28	" Kallu	67	58	160
29	" Mohd.Ahmed	62	65	164
30	" Abdula	56	64	160
31	" S.K.Pandita	56	66	169

Group B (Healthy Females)

32	Km.Parveen Salim	22	38	148
33	" J.Edward	23	42	152
34	" Malni	23	44	156
35	Dr.Suman Arya	25	45	152
36	" R.Prajapati	26	48	158
37	" Satwant Kaur	25	52	156
38	" Uma Rani	25	52	158
39	Km.Angilina Peter	25	44	156
40	" Manju Sundryal	22	45	156

S.No.	Name	Age (years)	Weight (kg.)	Height (cm.)
41	Ku.Vimi Katiyar	22	42	160
42	" Usha Singh	24	42	152
43	" Vimi Arora	25	42	154
44	Sis.Alexander	37	48	148
45	" Savitri D.	38	47	151
46	" Asha Robert	31	58	160
47	Smt.Kasoolan	46	64	158
48	" Kranti	45	66	156
49	" Leelawati	44	67	158
50	" Bimillah	53	50	150
51	" Afroz	56	54	148
	Group C (Ist degree relatives of patients of CAD)			
52	Dr.Alok Pandey	27	58	170
53	" O.N.Pandey	26	58	166
54	" Manglic C.K.	26	64	172
55	" Deepak Sharma	25	68	168
56	" Pawan Sood	26	85	180
57	Ku.Reena Austin	22	42	156
58	Dr.Arvind Saharan	24	74	172
59	" G.G.Sethi	27	74	172
60	Ku.Vinita Khanna	21	58	150
61	Mr.Ram Sewak	25	62	160
62	Dr.G.G.Singhal	27	70	168
	Group D (Diabetics Type I)			
63	Mr.Hari Ram	30	52	163
64	Mr.Ghan Shyam	26	51	160
65	Mr.Virendra Katyar	26	52	158
	(Diabetics Type II)			
66	Smt.Santosh	46	46	152
67	Mr.J.T.Karnani	67	64	160
68	Smt.Shakti	50	56	160
69	Mr.Narain Das	47	60	156
70	Mr.Abdul Jaffer	50	64	156

Group A (Healthy Males)

S.No.	TC			HDL			TG			VLDL				LDL		
	F	I	III	F	I	III	F	I	III	F	I	III		F	I	III
1	275	215	215	50	46	44	153	150	177	30.6	30	35.4	194	139	135	
2	250	130	140	47	39.6	41.1	113	103	158	22.6	20.6	31.6	180	69	67	
3	158	117	102	45	55	57	58	83	116	11	16.6	23.3	101	45	21	
4	163	153	179	42.5	40	42.5	125	133	141	25	26.6	28.2	95	86	108	
5	145	135	145	50	51.5	50	103	108	120	20.6	21.6	24	74	62	71	
6	155	150	160	50	46	50	120	136	158	24	27.2	31.6	81	76	78	
7	207	207	190	50	45.6	50	133	166	150		26.6	33.2	30	131	129	110
8	135	115	140	48	51.6	45	116	125	141	23.2	25	28.2	84	38	67	
9	210	165	250	37	37	43	145	162	200	29	32.4	40	136	95	167	
10	153	186	199	47	44	45	133	133	150	26.6	26.6	30.6	79	115	113	
11	117	148	143	37.5	36	37.5	100	108	125	20	21.6	25	60	90	80	
12	171	190	157	42.5	40	43.7	100	116	136	20	23.2	27.2	108	127	86	
13	199	216	194	52	52	50	200	220	210	40	45	42	105	118	102	
14	177	188	171	40	39	44	150	140	120	30	28	24	107	121	102	
15	165	180	155	39	47	39	170	174	180	34	35	36	92	98	80	
16	118	112	94	50	33	39	100	140	100	208	28	20	46	51	35	
17	112	88	118	41	38	40	130	116	120	26	23	24	45	27	54	
18	141	124	123	47	47	42	133	133	140	26.6	26.6	28	67	50	52	
19	147	136	154	37	39	40	133	183	166	26.6	36.6	33	83	60	81	
20	179	177	189	39	40	44	200	283	286	40	56.6	57	98	80	88	
21	177	216	210	56	60	58	160	165	180	32	33	36	88	123	116	
22	135	125	115	50	44	46	170	140	160	34	28	32	51	53	37	
23	195	160	168	45	46	35	200	200	183	40	40	36	110	74	96	
24	144	160	149	64	70	58	200	210	212	40	42	42.4	40	48	48	
25	249	138	155	37	40	37	190	210	230	30	42	46	81	56	71	
26	166	172	160	50	57.5	58	170	170	240	34	34	48	82	80	54	
27	130	142	148	54	54	61	183	166	120	36.6	33.2	24	39.4	54.8	63	
28	116	122	122	47	52	45	180	190	200	36	38	40	32	31	37	
29	160	144	155	50	45	50	100	100	183	20	20	36.6	90	79	68	
30	166	172	160	40	40	42	220	220	230	44	44	46	82	88	71	
31	183	166	155	41	45	37	180	200	190	36	40	38	106	81	86	

S.No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
32	127	122	133	52	57	52	96	100	103	19.3	20	20.6	55	44	60
33	146	131	140	57	59	62	154	130	163	36.8	27	32.4	40	45	45
34	128	122	121	50	55	55	145	148	156	30	29	27	40	38	39
35	164	176	170	52	55	80	141	130	153	28.2	26	30.6	83	95	89
36	188	164	152	62	66	66	145	149	136	28.8	27.8	27.2	96	61	59
37	213	222	182	56	67	52	150	159	162	30	31.8	32.4	127	123	97
38	176	164	140	69	57	56	144	117	145	29	23.5	29	78	83	55
39	221	197	221	71	69	70	210	220	220	42	44	44	108	84	107
40	168	166	166	22	33	50	171	179	179	34	36	36	127	97	80
41	188	172	188	28	33	40	182	128	214	36.4	25.6	42.8	124	113	105
42	155	122	133	18	26	28	128	192	214	25.6	38.4	42.8	114	57	62
43	195	150	155	35	34	41	162	196	256	38.4	39.2	51.2	83	77	62
44	206	220	248	56	62	63	150	150	152	30	30	30.4	120	128	154
45	254	250	248	67	62	65	266	300	298	53.2	60	59.6	133	127	123
46	177	174	209	42	45	62	260	233	280	52	46.6	56	82	82	90
47	250	230	245	39	30	33	299	321	356	59.8	64.2	71.2	151	135	140
48	198	215	198	44	43	50	250	254	280	50	50.8	56	104	121	102
49	225	230	235	41	41	40	256	270	276	51.2	54	55.2	133	135	140
50	154	171	177	41	33	26	206	239	283	41.2	46.6	56.6	72	91	94
51	196	200	204	42	40	38	246	260	270	49.2	52	54	104	103	112

Group C (First degree relatives of CAD)

S.No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
52	183	150	195	38.8	41.6	50	185	228	112	37	45.6	22.4	107.2	62.8	122.6
53	233	261	272	36.1	50.0	55.5	208	199	263	41.6	49.8	52.6	155.3	171.2	163.9
54	161	161	188	30.5	22.5	36.0	199	156	199	39.8	31.2	39.8	90.7	107.6	112.2
55	188	178	153	27.7	36.0	38	250	235	214	50	47	42.8	110	95	72.2
56	130	145	150	50.0	47	58	130	110	80	26	22	16	54	76	76
57	218	236	218	68.0	64	70	210	220	270	42	44	54	108	128	94
58	172	156	156	38.8	36	38.8	263	250	221	52.6	50	44.2	80.6	70	73
59	145	135	145	50	51.4	50.0	103	108	120	20.6	21.6	24	74.4	62	71
60	172	197	166	39	37	36	171	171	256	34.2	34.2	51.2	98.8	125.8	78.8
61	199	222	222	53	38	34	200	220	190	40	44	38	106	140	150
62	165	105	130	47	41.16	41.16	103	103	127	20.6	20.6	25.4	97.3	43.2	63.4

Group D (Diabeties)

63	135	145	175	53	41.5	50	144	120	120	28.8	24.0	24	53	79.5	101
64	125	135	150	33	36	27	150	160	220	30.0	32	44	62	67	79
65	155	165	188	48	47	43	160	160	200	32	32	40	75	86	105
66	309	268	262	57	59	59	331	425	350	62.2	85	70	185.8	124	133
67	225	230	190	41.6	41.8	30.5	256	321	235	512	64.2	47	132	124	112.5
68	261	220	214	36.7	36.7	50	188	195	244	37.6	39	48.8	166	124	115
69	205	215	205	43	44.5	41.6	208	220	274	41.6	44	54.8	120.4	126.5	108.6
70	240	205	260	44.5	47.2	44	198	214	250	39.6	42.8	50	160	115	166